JUNE.

CLINICAL LECTURES

ON THE

DISEASES OF OLD AGE

BY

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NOTE.

Lectures I. to XXI. inclusive, with the *Introduction*, are those delivered by Prof. Charcot; XXII. to XXXII., inclusive, by Prof. Loomis.

The *Introduction* and the first eighteen Lectures correspond with the numbering in the original, while XIX., XX., and XXI., are I., II., and III., respectively, of the *Appendix*, and are thus renumbered to avoid confusion.

Prof. Loomis' Lectures are made to commence with number XXII. for the same reason.

L. H. H.

PREFACE.

THE few Lectures which I have been requested to add to those delivered by Professor Charcot on Diseases of Old Age, embody, in the main, the salient points in Diseases of Advanced Life which I have been accustomed to impress upon my classes at my clinic in Bellevue Hospital.

They are presented in this form with the hope that they may prove acceptable additions to the very interesting lectures of Professor Charcot.

ALFRED L. LOOMIS.

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[1] Lectures delivered by J. M. Charcot, in the Salpêtrière. 1867. (Lectures xix., xx., and xxi., are i., ii., and iii. of the Appendix, renumbered to avoid confusion.—L. H. H.)

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CLINICAL LECTURES

ON THE

DISEASES OF OLD AGE.

INTRODUCTION.

EMPIRICAL AND SCIENTIFIC MEDICINE—A COMPARISON BETWEEN THE ANCIENTS AND THE MODERNS.

GENTLEMEN :—The course which we to-day inaugurate is purposed to acquaint you with the general characteristics that distinguish the pathology of old age from that of adults, and to fix your attention on some of those diseases which are more especially met with in asylums reserved for the aged.

Within as close limits as possible I shall endeavor to proceed by a method which is suitable to theoretical instruction. We shall not, however, neglect the clinical aspect ; and I hope, indeed, to be able to show you, at the bedside, some of the types which are to be the basis of our descriptions.

You are now acquainted with the object of these studies ; and we can at once commence with the subject, for I am of Condillac's opinion, and I think that considerations general in their nature are much better inserted at the end of a course than at its beginning.

Still, there is a classical tradition, as it were, which puts a professor under the obligation to explain himself at the very commencement in a more or less categorical manner upon certain fundamental points, and, after a fashion, to make a profession of his scientific belief.

I do not think I ought to exempt myself from this obligation, and shall courageously enter upon the accomplishment of this task—often thankless, always difficult—reckoning not a little upon your indulgence and upon the kindness which you have so often shown to me.

There is a simple, and, so to speak, natural means of broaching these great and important questions : it is to inquire as to how, in the course of the progressive development of scientific culture, they have become known and how they have been settled ; in this way history becomes a means of criticism.

Now, in following that path, it will soon become very clear to you that pure observation was never attained at any epoch without supreme efforts having been made to prevail over the spirit of hypothesis.

As for that which especially concerns medicine—and with this we are alone occupied here—even the most stoical minds have never confined themselves to the mere statement of facts, but have sought to make a common bond of union between them by means of some theory.

From the very commencement we see the mind of man occupied as much, nay more, with the subjective relationship of things than with their reality; the data furnished empirically by observation, when still but recently obtained, are brought together in a class—tested, one by the others, in order that systems or theories may be derived from them.[1]

In this, it must be confessed, lies one of the necessities of the human mind, and, according to a celebrated saying of Kant's, it seems that our ideas are forced to cast themselves in this common mould; and it was precisely this which was recognized by the founder of positive philosophy himself, who will undoubtedly be reproached by no one for having opened the way for hypothesis, when he declared that, even if a theory is to be founded entirely upon observation, it must still from sheer necessity be guided by some theory in order that it may yield to observation a profitable result.

The real wants of our minds can, however, always be distinguished, in this connection, from the manifold exaggerations with which systematic thinkers allow themselves to become involved. Hence we see in reality the existence of a method of speculation which is grounded strictly upon facts, as well as the existence of a method of observation keeping aloof as much as possible from premature speculation; and the whole question consequently resolves itself into the discovery of a common ground whereon these two methods can meet.

Gentlemen, viewing matters from the point which we occupy to-day, I think that we can discover a radical difference between ancient and modern medicine.

The first has always been deficient in those elements necessary to the construction of a positive theory, and for this reason the numerous attempts which it has made in that direction have of necessity signally failed. Modern medicine, on the other hand, possesses some of the material that could serve such a construction; but, profiting by the mistake made by its predecessors, it knows above all what paths should remain impervious to speculation, and it likewise knows what tracts it may survey without falling into error.

I shall endeavor to develop this theme in the course of the present lecture; but, first of all, we must be acquainted with that time in history when the old system of medicine ended to give way to the new.

I.—In our day a profound, a radical revolution has occurred in medicine.

It would undoubtedly be necessary to go back to far distant times in order to discover the first origin—the point of departure—of this mighty change, whose influence we still feel to this day. But the direction of this movement has been distinctly marked out, and its aim has been most brilliantly set forth, only toward the end of the eighteenth and the beginning of this century.

It would be going quite too far if we were to say that an unfathomable abyss opened just at that period and separated the medicine of ancient from that of modern times. No; traditional ties are not sundered; the labor of times gone by is not lost; and we shall treasure up the immense

[1] Dechambre: Introduction to the Dictionnaire Encyclopédique des Sciences Médicales, p. xix.

heritage which our predecessors accumulated in the course of centuries. Still, it must be confessed that new horizons have opened to us, and that the views of modern science are from a standpoint which has risen as it has altered.

The forerunners of this reformation were Vesalius, Harvey, Morgagni, and Bichat, the creator of general anatomy. Corvisart, Laennec, and Broussais co-operated to a very great extent in the movement; and now come our immediate teachers, and it is with pleasure that I ask you to observe that all these names are names of Frenchmen.

It appears that the marvellous progress made by experimental physiology in these later times, under the influence of Müller, Claude Bernard, Longet, and Brown-Séquard, has finally brought over even the tardy and the timid, and settled the question.

The essential characteristic of this revolution consists in the direct, the immediate—and we will say legitimate—intervention of anatomy and physiology in the domain of pathology.

Before the period when this revolution occurred, medicine was almost wholly restricted to the study of symptoms; after that time it became, in succession, anatomical, then physiological, and next acquired decided scientific tendencies.

The fundamental principles accepted by modern medicine can, I think, be ascribed to two characteristic features.

In the *first* place, symptoms which used to be considered in an abstract manner, and, to a certain extent, apart from our organism, are to-day intimately connected with it; according to Bichat's precept, the seat of the malady should be sought for.

The inflexible logic of Broussais forced him to proclaim that he knew of no derangement of function without a corresponding lesion of an organ (we should rather say *tissue* to-day); symptoms, then, are in reality nothing but the cry from suffering organs.

In the *second* place, it was possible for the ancients to consider disease as a thing independent of the organism—as a sort of parasite clinging to the economy; while to-day—and again to Broussais we owe it for having succinctly asserted the principle—it is merely a disturbance of the inherent properties of our organs. It is here no question concerning the appearance of new laws, but only of the perversion or derangement of pre-existing ones.

We recognize the enormous part which the sciences of anatomy and physiology are henceforth to play in the interpretation of morbid phenomena; but is that saying that these two branches are to absorb pathology and rule it with an absolute hand?

Gentlemen, a serious difficulty, demanding discussion, presents itself at this point, and I ask your permission to enlarge somewhat thereupon.

It is absolutely certain that pathology can be greatly illumined by physiology, but it is just as certain that it cannot be deduced from physiology.

Allow me in this connection to recall to your minds a profound thought which is found in Hippocrates: "Who could have foretold," says he, "from the structure of the brain, that wine could derange its functions?" M. Littré, who quotes this passage, adds the question: "Who could have been taught, by his knowledge of the human body, that the *emanations* from marshes would cause intermittent fever?" It is, as you see, a complex problem. Really, in addition to the physiological condition supposed to be known, it is likewise necessary to search for the manner, which is in-

fluenced by the action of the morbific cause. In other words, in order to know what will be the effects of any morbific agent upon a healthy organism, it is indispensably necessary that experiments shall have been made.

Thus you see that pathology, in the presence of anatomy and physiology, has the right to preserve a certain degree of independence, and to possess a certain amount of autonomy. A pure and simple statement of pathological facts evidently furnishes us with data of the highest importance, needing no support other than that they furnish of themselves, and these, so to speak, constitute the material of medicine—a *materia medicinæ*. The intervention here of biological sciences is without doubt indispensable, including, too, the physico-chemical branches which are connected with biology; and in default of their support, our researches would very often be made at random, as if we groped in the dark;[1] still, their part, however deeply pathological observation is concerned, is, in a certain measure, a subordinate one; they are only the means, not the end. The observer confines, and wishes to confine himself to the recording of facts and to the consideration of them in their natural relationship. Here, I believe, lies the true character of empirical medicine, using "empirical" in all the dignity of its etymological signification. On the whole, we may designate by that title the science of medicine when viewed in a still imperfect state, and in a period when facts were not united among themselves by a physiological theory.[2]

Requin says: "The day when science shall have attained a complete knowledge of normal man, to the very depths and inmost parts of his organization, and into the most secret mysteries of his life; the day when science shall have unveiled all the secrets of the pathological condition, and understood every modification that external agents can produce in the economy—that day science will be completed."[3] But, at this present hour, reality is far, very far, from approaching this ideal; pathology, physiology, anatomy itself, all are in process of evolution.

Hence, I think that in a correct method it is well that the two aspects that have been mentioned, as well as their provisional titles, should remain systematically separated, while their conjunction is, at the same time, favored as strongly as possible and justifiable. To sum up, then, we must avoid, as Bordeu says, a forced and unwilling union between dogma and empiricism.

With these reservations we are the first to recognize the immense service rendered to pathology by the well-directed application of modern physiology, and to boldly assert that in it lies the future of medicine.

And besides, our predecessors did not lack a feeling of the importance which ought to be accorded to physiology in the study of medicine, but they lacked more exact and extensive ideas concerning the difficult problems which they sometimes endeavored to solve without having first determined their profoundness. It can even be affirmed that at all times men have striven to interpret pathological facts in accordance with the views of the physiology then in vogue.

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[1] "Clinical observations made in our times are superior to those gathered in past centuries only because of the superiority of the moderns over the ancients in the matter of anatomical and physiological science." (Ch. Robin: Journal de l'Anatomie, etc., 1867, p. 297.)

[2] Littré: Dict. de la Langue Française, art. Empirisme.

[3] Boerhaave says: "Qui itaque haberet perfecte intellectas omnes conditiones requisitas ad actiones, ille perspiceret clare defectum conditionis ex cognito morbo, et rursus bene caperet ex cognito defectu naturam morbi inde necessario sequentis."

Just here allow me to repeat to you the very words of Daremberg, who gives his opinion on this point with all the authority which a thorough knowledge of history imparts. He says: "Nothing is better than a good physiology, or at least a physiology which, grounded on experience, still carries within itself inexhaustible germs of perfection. Such a physiology reforms medicine and transforms therapeutics; but, again, nothing is more disastrous, more opposed to the progress of pathology than an unsound physiology, than an "*à priori*" physiology, which daily finds in itself the best reasons for plunging deeper and deeper into obscurity, and restraining the flight of science. It was in vain that the most delicate and difficult observations multiplied in the Hippocratic school at Alexandria; ideas are more stubborn than facts. Physiology resists so bravely, that it misconstrues the discoveries of anatomy and the conquests of pathology, in order to bring them under its laws. Very different was it when beneath the hand of Galen the experimental method, already touched on at the Alexandrian school, transformed the physiology of the nervous system; then the whole aspect of medicine changed."[1]

Now, gentlemen, that "*à priori*" physiology alone is responsible for those too notorious systems which, at various revivals, have excited such a baleful influence upon the development of science—Naturalism, Stahlism—[2] and all the forms of ancient and modern vitalism, not excepting that of Bichat, touch it on the one side; and iatro-mechanism[3] and iatro-chemistry,[4] on the other.

The former take away the principles of life from the organism, so that they may govern it as would a capricious ruler; while the others daringly attempt to give a physical or chemical interpretation to life, and this, too, at an epoch when the real relation of biology to the physical sciences was not even surmised. Happily, such errors are impossible in our times. Placed upon a solid basis, physiology can no longer be dangerous to medicine; on the contrary, it has become its most solid support. It encloses it on every side, if I may use the expression, in a net-work with compact meshes, no longer permitting rash speculations to pass through it; and even if, in our times, errors are yet committed in some instances, they only evince a faulty application of the method, and not an error in its principles.

Wise and well-balanced thinkers have always energetically protested against usurpation by an unsound physiology; and from this standard we must look with admiration upon those physicians who were devoted, on principle, to the cultivation of pure observation, from among whom towers the mighty form of Hippocrates in the foremost rank.

Gentlemen, what are the products of that medicine systematically isolated from all extraneous contact, and consequently reduced to its own forces, and founded upon pure observation? What is its character, how much may it claim, where does its province end? Here are questions that certainly deserve to be examined. History offers in this respect a series of experiments made for us, prolonged over the several centuries, and whose results are to-day offered for our estimation of them.

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[1] Résumé de l'Histoire de la Médecine, etc.: Union Médicale, 1865.

[2] Stahlism: the "phlogiston theory."—L. H. H.

[3] Iatro-mechanism: a system of treating diseases by the application of mechanical forces.—L. H. H

[4] Iatro-chemistry: a system where diseases were treated with chemical preparations.—L. H. H.

II.—But this leads us back into the very midst of antiquity, to the best days of Greece—to the time of Pericles.[1]

In this epoch anatomy was in a most rudimentary condition ; the nervous system had not as yet been distinguished from the tendinous structures. The brain was considered a gland. They thought that the arteries were filled with air, and the real distribution of the venous system was wholly unknown.

Physiology was even below this level ; it was grounded only upon fantastical notions ; it had not even disengaged itself from the speculative philosophy of that period, which, in its audacious aspiration for immediate knowledge of the universe, ignored a patient and modest search for facts, and even thought this almost unworthy of a learned man. For the radical error of Greek philosophy, as Herschell says,[2] was to imagine that a method which had produced such dazzling results in mathematics could also be applied to objective reality, and that, if the most elementary ideas or the most evident axioms were departed from, everything was in dispute.

In the midst of these circumstances, apparently so unfavorable, we see the school of Cos[3] spring up. To us it seems that all its criticisms were directed against the Cnidian[4] physicians, who did not know how to resist the philosophy of the time as did the former school. It insisted that medicine should proceed along its own way, resting upon fact and not upon hypothesis ; and in this way it erected the edifice which deserves the admiration of every age.

It can be compared to the *chefs-d'œuvre* of Grecian sculpture executed by masters who had only observed external forms, but whose beauty we have not yet learned to surpass, with all our anatomical knowledge.

Hippocrates considers the body as a whole, and, if I may say so, in its outlines, without pretending to penetrate the internal mechanism.

He thus views it in its relations with the external world, in order to ascertain the modifications which it undergoes from it.

This last standpoint leads up to a vast and comprehensive etiology, where the influence of seasons, of climates, and, among individual causes, that of age, stand out in bold relief.

As the year passes successively through the hot and the cold, through the dry and the wet seasons, the human body undergoes changes, and diseases assume certain characteristics. Upon this foundation it established the doctrine of medical constitutions corresponding to particular atmospheric conditions, a doctrine which still exists in our day despite the exaggerations which it has suffered, because at bottom it has truth in it.

Climate, as it were, is one permanent season, and one whose effects are more marked since its influence is ever acting. If the Greeks are brave and free, if the Asiatics are effeminate and slaves, that comes from the climate in which these people live. In this idea, as you see, is the germ of the geographical fatalism—a doctrine which has been developed in modern times by Herder, and one which a literary philosopher of to-day, M. Taine, worked up with so much skill under the title of the "theory of environment" (Théorie des milieux). Here there is a flagrant exaggeration ; but who can question the influence of climate upon the habitual state of body

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[1] In this passage it will be easy to recognize what has been borrowed from the excellent explanatory introduction that M. Littré has prefixed to the works of Hippocrates.

[2] Discourse on L'Étude de la Philosophie Naturelle, p. 134.

[3] Cos, an island in the Ægean sea ; the birthplace of Hippocrates.—L. H. H.

[4] Cnidus, an island near Cos.—L. H. H.

and mind, and above all, upon diseases which may affect us? Following out this analogy, Hippocrates looked upon ages as the seasons of life, and consequently attributed special diseases to them. Without accepting this interpretation, modern science has still fully confirmed the truth of the fact, and we know that there are diseases which pertain to the different periods of life.

That which attracts us at the onset and impresses us most strongly in the Hippocratic pathology, is the importance he accords the general condition, and the indifference he exhibits with regard to the local state of affairs. This point of view will appear singularly exclusive unless we remember the conditions under which Hippocrates made his observations. He concentrated his attention pre-eminently upon acute febrile diseases, and, as M. Littré has remarked, without doubt more especially upon the remittent and pseudo-continued fevers which are as prevalent in Greece to-day as they were in the time of Hippocrates. Now, it is a recognized fact that fever almost always involves the same state of the system, independent of the causes which produce it and the different forms it may assume. But that which, according to Hippocrates, was of prime importance, consisted in discovering certain signs which enabled crises, symptoms, and the termination of the disease to be foreseen, and which furnished indications for the exhibition of remedies.

It was to attain this end that he attentively examined the features and the color of the face, the attitude of the patient, the heat of the body, the respiratory movements, the urine, sweat, and the various evacuations. Is it not with almost the same end in view that we try to find out the state of the forces, the amount of arterial tension, and the oscillations of the central temperature, when the local condition does not furnish useful indications?

And the theory of crises, even after enduring the stern test of modern criticism, has still survived, though stripped of the intentional character ascribed to it by the ancients.

You see, gentlemen, from this rapid outline of the pathology of Hippocrates, what results the method of observation may lead to; the facts gathered by it, and views it has proved, resist the action of time. They have reached us without having lost any of their striking truth. And we further state that, without transgressing its legitimate prerogative, this method may suggest general views and furnish data of a superior order.

If in reality there exist an observation especially analytical and limited to detailed facts, in which the moderns have noticeably excelled, there yet exists observation on a grand scale, more familiar perhaps among the ancients—one which is not confined to the examination of isolated phenomena, but which views them, on the contrary, in their mutual relationships, in their order of succession; such, definitively, as nature offers to him who knows how to view things from a somewhat elevated standpoint.

Thus we have come to recognize that every disease has its own evolution, a special manner of development, a peculiar series of symptoms, which allow a description to be made of it after a common type in the midst of variable, accessory circumstances.

Thence has issued the idea of unities or morbid species, a notion perfectly correct since it corresponds to a fact of experience, but the signification of which has been strangely altered when things have gone so far that diseases are regarded as concrete beings like individuals, by the same right as an animal or a plant.

We know now that a disease sometimes runs rapidly through the suc-

cessive stages which are part of its natural history, to reach its termination; and we know that sometimes, on the other hand, it requires a long space of time to pass through its various phases of development. Thus arises between acute and chronic affections a distinction which evidently answers to the results of clinical observation, but which ought not to be looked upon as a line of absolute demarcation, since the acute and chronic form of one and the same pathological condition often merge into each other by imperceptible transitions.

We have further seen, by observations which have multiplied, that various morbid states coexist or succeed one another in the same person or in the same family, following a definite order and after certain laws; and hence it has been concluded, by a very simple process of reasoning, or rather by direct intuition, that those diseases were not isolated, and that they must unite in a common cause which served for their bond. In this way the grand conception of constitutional diseases and diatheses was produced, and this idea originated in pure observation. Hypothesis does not commence until the moment when, in order to attribute existence to this unknown cause, the mind, following the bent of the day, has now ascribed an influence from the nervous system, and now a modification of the crasis of the humors, or the presence in the blood of some morbific material.

In the course of time, analytical has succeeded synthetical observation, which has not, moreover, been always neglected. And thus, as the world has grown older, science has been successively enriched by an immense number of general or partial facts, which positively constitute the only traditional medicine that commands respect.

We have now arrived at the limit of this rapid review. The examples I have given are sufficient for us to appreciate the resources of the method of observation applied to medicine, even when it voluntarily confines itself to the sphere of the external phenomena of disease, that is to say, the symptoms.

But it is easy to recognize at the same time that a pathology thus deprived of its natural support can amount to nothing, even in the most skilful hands, but a more or less elevated empiricism. It is that which undoubtedly constitutes the common, the fundamental course of all scientific construction, and it has even been possible to establish upon such a basis a practical medicine which nothing hinders from arriving at a certain degree of perfection.

But the work remains incomplete, and the human mind, urged forward by an irresistible force, is not able to stop by the way.

The efforts it has made to finish its task have of necessity been futile so long as anatomy and physiology were not established. And this should make us indulgent toward those systems which have successively arisen from antiquity to our days. But at last the time has come, reform is accomplished, and we may now appreciate what fruit it has produced and foresee what it will bring forth in the future.

III.—Clearing with a single bound a period of two thousand years, we are now to confront the results of pure observation with the work of the modern mind, armed with all the means of investigation which it to-day possesses. And to render the contrast more striking, we shall pass over in silence the transition epochs so as not to be busied with anything except the scientific movement properly belonging to this, our century.

You have, gentlemen, no doubt heard mention several times of a new pathological anatomy, as opposed commonly to the pathological anatomy

of the ancients. It is proper that we should at the very onset distinguish what each expression signifies.

I shall speak just here of an historical point to which I ought to direct your attention for a moment: it is quite recent history, for it is not so long since pathological anatomy asserted itself for the first time as a special branch of science. One of the first attempts at systematization which became law, dates from 1812. The statement of the doctrine—its code, as it were—is found recorded in one of the first articles of the great "Dictionnaire des Sciences Médicales."

This document, precious on so many accounts, bears the signature of two illustrious men—Bayle and Laennec.

I wish to attempt, gentlemen, to characterize in a few words that first-named pathological anatomy which has for half a century held undisputed sway.

Its means of investigation are very simple: it takes the word anatomy in the rigorous exactitude of its etymological sense, and knows nothing besides save the scalpel.

For it, the alterations occurring in organs can be determined only after changes have taken place in their *relations, volume, consistency, density, color,* and *general aspect.*

It is impossible, as a general rule, to look for characteristics of modifications in *texture,* since this is almost always ignored.

What efforts must be made in order to establish the identity of an anatomical type with the aid of such data? But the masters surpassed themselves, and produced in this manner more than one inimitable model. In spite of all things, every one understands how difficult it is to fix in the mind such fugitive traits as those, for example, taken from the *aspect* and the *color.* These have shades and contrasts which the most picturesque language finds it difficult to express. Thus the anatomo-pathologist has many times felt the need of becoming an artist, or of invoking the aid of the stranger's pencil. It is quite natural that art should intervene here, where the figurative side has such great importance. To its co-operation we owe more than one precious collection, and, above all else, the imperishable monument erected by Professor Cruveilhier, under the name of "Atlas d'Anatomie Pathologique du Corps Humain."

From the commencement it was foreseen that a day would come when all the lesions that the eye could not perceive would be definitely described, classified, and catalogued. It was thought that then the work would be finished, for it was not known from what side improvement could come. That is undoubtedly why the heads of the school did not dream that their science would play any but a very modest rôle in the whole sphere of pathological science. They even at all times endeavored to circumscribe it within narrow limits, as if they wished, by this means, to preserve it from all the allurements which might have compromised it.

In the first place they hasten to declare that their anatomy is applicable only to a limited number of diseases, since many of them do not possess lesions which are appreciable by its means of investigation.

And further, even in the domain of organic diseases, where it would seem right for it to obtrude, its part is still not a very ambitious one. Indeed, the object is, above all, to enrich nosology with new lights, allowing discrimination between diseases the analogy of whose symptoms might have led to their confusing. The distinction being stated, the end was attained; for pathological anatomy voluntarily limits itself—and on that point is fully resolved—to the viewing of lesions by themselves, independent of the symp-

toms which accompany them. It is not necessary to look for any explanation here concerning the proximate cause of disease, or the mechanism of its formation.

Such, gentlemen, if I do not mistake them, are the most original features of this pathological anatomy, that has sometimes been ironically called "*the dead anatomy.*" I have not exaggerated any; I believe that at least I have faithfully portrayed the tendencies of the first masters.

Their successors or imitators have, in truth, very often transgressed the limits which they imposed upon themselves, and it is still easy to follow the tracks of their brilliant incursions into more than one region believed to be closed to pathological anatomy.

The method stood, however, at least in a general way; but the moment was near when it was to undergo a radical reformation.

You are acquainted, gentlemen, with the profound changes which *the anatomical school*—for by this title is it designated—has worked in medicine, with those limited and circumscribed functions which it assigned to itself. This is a point upon which it is unnecessary to dwell. I only wish to show that, even to this day, notwithstanding so much progress has been made in various directions, it has in a great measure maintained its teachings in all their *authority* and all their effectiveness. In this way studies by the naked eye, such as it has instituted, and like the autopsy whose regulations it made, will always constitute the first operation through which all regular investigation must pass.

From such considerations as these it is easy to foresee that the new anatomy can be called upon to complete the anatomy of the ancients, and to expand and develop it, but that it ought never to be substituted for it. Gentlemen, if I were to characterize in a word all the service rendered us by the first pathological anatomy, whose creation can be counted among the glorious events of our country, I would say that it has taught the physician *to think anatomically.*

When circumstances, already prepared for a long time, made reform and evolution possible, it took its first step in the operation by means of a wholly renovated physiology and one which had decidedly entered the experimental channel—the physiology of Magendie and Legallois. It was then definitely fulfilled by the intervention of histology armed with the microscope.

Then men saw that it was not only the dead organ, the one such as an autopsy exhibits, with which the physician should be acquainted; but that it was the living organ in action, exercising its proper functions, and modified by the morbid condition which must be reconstructed in the light of physiological ideas. The organ under his eyes represents only one of the phases, often the last, of a morbid process whose course he must trace back in order to penetrate to the very first effects of the morbific cause.

Here is the outline of the physiological programme; but you yourselves will confess that it would have still remained a sealed book for a long time were it not for the co-operation of histology.

Thus, as we have seen, the anatomist from principle stops, as a matter of course, at the surface of an organ. If at times the microscope was employed in the study of lesions, it was not guided in that kind of investigation by any systematic idea. Now, gentlemen, in histology the microscope alone is not simply to be considered; there is more than this, there is a system, a doctrine. Here is the essence of what constituted this doctrine: *analysis must penetrate to the depths of an organ, to the very elements, or elementary anatomical constituents.*

It is in these elements that the conditions of incomplete life may be truly said to reside; an organ is but an aggregate, a collective, a resultant; and hence, by the aid of the microscope and reagents, we must penetrate to the elements in order to get at the modifications which they undergo from the influence of morbific causes, and from these modifications to deduce the reason of the derangement produced in the whole organ.

Thus, gentlemen, pathological histology, from which histo-chemistry is inseparable, has not its sole aim in a limitation of the number of non-material diseases, by showing that lesions may be present where the eye cannot perceive them; in furnishing new means of diagnosis, new nosological characteristics; neither is its part restricted to the unveiling of the hidden reason of anatomo-pathological forms, recorded and classified by microscopy, and to affix a more lasting and scientific characteristic to them, by ascribing each one to its corresponding modification in the texture of the organ; its aims stretch still farther, for it also has for an object a kind of intimate pathological physiology, which, as it were, follows step by step the various phases of the morbid process in each elementary part, striving to avail itself of the least transition which binds the pathological to the normal state.

On this side you see that *pathologico-histological anatomy* touches on pathogenesis, or rather mingles with it, and at the same time also allies itself to physiology, which in this special direction takes the name of pathological physiology.

Now, gentlemen, it is important to notice—for it is a characteristic feature—that the end which pathological anatomy proposes to itself cannot be reached without establishing an incessant approximation of the lesion that is studied even to the minutest details of its development, and the pathological circumstances minutely observed at the bedside. And in this manner, as a consequent obligation, as a logical necessity, pathological anatomy, in proportion as it penetrates more deeply into the intimacy of tissues, becomes simultaneously more animated and living, tending toward a closer union with the clinical.

However, gentlemen, in the face of a manifest functional derangement whose seat is indicated by physiology, histological analysis often remains even yet powerless to state what material vice corresponds to the disturbance of function. Is that saying that we must resign ourselves to the belief, contrary to all analogy, that a functional lesion, a derangement of organic properties, can exist without any corresponding material modification? As for myself, were the question urged upon me, I should not hesitate to place myself by the side of Reil, Broussais, Gorget, and many other eminent minds; I would recollect that, even in the normal performance of the functions of life, the labor of the organs is not accomplished without a material change taking place—a correlative destruction and reparation. Every function tends to destroy the instruments by means of which it is performed. Muscle in a state of repose has an alkaline reaction; it becomes acid when it is tired.

An analogous modification has been observed in the nerves and the spinal marrow, and even the cerebral substance itself is, according to Heynsius and Funke, acid when the individual is awake, and alkaline during sleep. Such examples as these show us that in the anatomical elements the maintenance of life is closely bound to the existence of an incessant molecular work which manifests itself by appreciable chemical phenomena. Is it not easy to conceive the existence of lesions of elements which, without altering their form, hinder that organic movement in the absence of which their

functions cannot be performed? Without altering their form, carbonic oxide robs the red blood-corpuscles of their power of absorbing oxygen. Henceforth they roll along the torrent of the circulation, like so much inert dust; and were it not for the change in color which they present, nothing external would betray the profound change they have undergone. Cannot an analogous alteration in the blood-globules be at least suspected in certain grave conditions of the organism, when intense dyspnœa appears, without there being pulmonary or cardiac lesions?

In those patients who rapidly succumb in the course of acute articular rheumatism, the blood and the serous contents of the pericardium and the synovial sacs often have an acid reaction. In the case of those unfortunates who are struck by lightning, anatomy can often reveal no appreciable lesion; yet cadaveric rigidity here supervenes almost instantly after death, continues scarcely a moment, and almost immediately gives way to rapid putrefaction. The same thing happens with over-driven animals, and these same phenomena occur, as Brown-Séquard has shown, in a limb whose vital properties have been exhausted by the prolonged application of an electric current.

Is it not almost certain that all these changes, which are ascertained only after death, correspond to material alterations which already existed during life, but which hitherto have remained inaccessible to our means of investigation? But I shall not insist upon this point; let it suffice that I have conducted you to a territory which is still almost unexplored, and which promises abundant harvests in the future.

IV. Gentlemen, it remains for us to determine the relations which ought actually to exist between pathology and physiology.

The speculative or contemplative physiology of by-gone times will not be considered here, but rather the science which has been constructed for us by the great teachers of modern times, successors of Haller, Legallois, and Magendie.

While fully recognizing that living beings present phenomena which are not exhibited in the dead body, and which, consequently, peculiarly belong to them, the new physiology still absolutely refuses to look upon life as a mysterious and supernatural influence which acts as its caprice dictates, freeing itself from all law.

It even goes so far as to believe that vital properties will some day be brought over to properties of a physical order; it states, at least, that from this moment one must not establish an antagonism, but a correlation between these two orders of forces.

It purposes to bring all the vital manifestations of a complex organism to workings of certain apparatuses, and the action of the latter to the properties of certain tissues of certain well-defined elements.

It does not seek to find out the essence or the *why* of things, for experience has proved that the human mind can never pass beyond the proximate causes, or the conditions of phenomena's existence.

It recognizes that, in this regard, the limits of our knowledge are the same in biology as in physics and chemistry. It remembers that beyond a certain point, nature, as Bacon says, becomes deaf to our questions and no longer gives an answer.

Further, it makes no pretensions to dominate medicine, but only to enlighten and furnish a solid basis for its speculations.

It brings to it, in reality, a method long since tested, the experimental method, that admirable instrument which in its hands has already unveiled so many mysteries.

Thus, M. Claude Bernard, a great teacher in physiology, expresses himself in various places of his remarkable work, with which you are all acquainted, and where, in a profound manner, he discusses the question with which we are here occupied.[1]

I shall limit myself to bring forth the express condition which he also imposes upon all legitimate intervention of physiology in the domain of medicine. First, pathological interpretation; such, says Bernard, is the imprescriptible rule; to first state the morbid phenomena, and then to endeavor to explain them from the standpoint of physiology, when this is possible in the actual condition of science.

The opposite procedure, which consists in starting from anatomy and physiology, in order to deduce the conditions of the disease, is full of peril and bristling with danger. Let us not be seduced by the elegant view, the ingenious ideas which it can suggest, for experience proves that too often it has led to an imaginary pathology corresponding in nowise to the reality of things.

It appears useless for me, gentlemen, to dwell longer upon the innumerable services already rendered medicine by the intervention of the data of modern physiology.[2]

I wish, however, to dwell for a moment upon what it is agreed to call experimental pathology.

The mutilations to which the physiologist subjects an animal, in order to penetrate the mechanisms of normal actions, are almost always attended with various morbid disturbances which may already become a subject for thought to the physician. But experimentation may apply itself still more directly to the problems which pathology offers to it. We have really succeeded in creating various morbid conditions in animals, either in causing them to suffer certain mutilations, or in submitting them to the action of poisons, viruses, venoms, putrid materials, etc.

The idea of producing, in this manner, artificial diseases, is far from being a new one, and we must go back to Lower, Baglivi, Van Swieten, and Autenrieth, in order to find the first traces of it. But, above all, it is in these last years, and from the impulse given to it by Magendie, that experimental pathology has really been constituted and assumed all its scope. The works of Claude Bernard, Longet, Brown-Séquard, Virchow, Traube, Vulpian, and many others, are ready to testify what can be attained by this method. Nevertheless, the brilliant results it has already furnished in its short career should not allow us to forget that certain limits seem to be imposed upon it. Experimentation succeeds in producing a temporary glycosuria by the aid of a traumatic lesion. It reproduces marvellously well the various symptoms of thrombosis and embolism. It accelerates or stops at will the movements of the heart. It induces at its pleasure all the symptoms of uræmia. Thanks to certain lesions of the nervous system, it originates pleurisy, pneumonia, and acute pericarditis, more or less comparable with those observed in man. Recently it succeeded in developing in an animal the phenomena of traumatic fever, by injecting into its blood the liquid of another animal from the surface of a recent wound; and thus was confirmed an idea promulgated a long time ago by a French surgeon.

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[1] Introduction à l'Étude de la Médecine Expérimentale. Paris, 1865. Especially consult pp. 117, 119, 125, 127, 140, 336, 343, 347, 348, 358, and 369.

[2] Consult, upon this subject, the remarkable discourse delivered by my friend, Dr. Brown-Séquard, before the College of Physicians of Ireland, February 3, 1865: On the Importance of the Application of Physiology to the Practice of Medicine and Surgery. In the Dublin Quarterly Review of Med. Sci., May, 1865.

But those diseases which have a slow process of evolution can be said to escape it most often.

Constitutional diseases and diatheses, in particular, seem to be inaccessible to it ;[1] and how can it be otherwise if the conditions of development of these diseases are those with which the physician himself is oftenest unacquainted? Let us not forget, however, to point out the analogies existing, in some respects, between those diathetic diseases called spontaneous, and certain slow intoxications, such as saturnismus and chronic alcoholismus, which have been successfully reproduced in animals in imitation of the corresponding affections in man. Recollect also the remarkable experiments by the aid of which my illustrious friend, Brown-Séquard, produced in the guinea-pig a kind of epilepsy transmissible by means of heredity.

However it may be, if experimental pathology is unable to originate certain diseases in their entirety, it can nevertheless often imitate the symptoms thereof and cause them to appear separately, one by one, if not always in the regular order of their natural succession. Thus, it can facilitate the study of certain general questions, such as impressionability, local predisposition, and also enlighten the mechanism of the generalization of morbid conditions. Finally, we owe to it that fine analytical study of poisons, whose results already make us foresee the approaching construction of a truly rational therapeutics.

And now, gentlemen, it would be a very easy matter to prove to you that, by combining clinical data with those furnished us by pathological anatomy, pathological histology, and physiological experimentation, we frequently arrive at a perfectly rational and almost complete conception of certain morbid conditions. But time presses, and I must therefore conclude.

V.—The comparison I have endeavored to outline between the pure observation of the ancients and the methods of the moderns has led to conclusions which must now be determined.

First.—I think I have established the fact that the empirical method is the necessary gateway of the science. We shall never be able to abandon that method which has been tested by the experience of centuries. It should always stand boldly forth in order to control, and to act as the counterpoise of scientific speculation.

Second.—But there is one side where theoretical views can and ought to be legitimately introduced into pathology. Clinical observation, perfected by the intervention of new operations, should ally itself with the general sciences and approach nearer and nearer to physiology so as to originate a truly rational medicine.

Such is the end towards which we should always progress ; but we must advance along this path with prudence, and never abandon ourselves to premature generalization ; that would be compromising the future of this grand movement of renovation in which we are to-day participating.

But, some one will ask, what is the good of all these fine words and these grand notions? Have these scientific tendencies about which there is so much ado had a favorable influence upon the practice of the art? Are more sick cured to-day than in by-gone times?

This, gentlemen, is a very indiscreet question—one which, truly, will be but too easily turned against our adversaries. We shall content ourselves

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[1] See G. Sée : Leçons de Pathologie Expérimentale, p. 11. Paris, 1866.

with answering, in the words of an honored teacher, that art without science very soon degenerates into routine.[1]

Commonplace scepticism, which is so readily opposed to all progress of the human mind, is a convenient pillow for lazy heads; but in this epoch there is not time to go to sleep.

To place in its true light the favorable influence which, in another way, scientific tendencies might exert upon the advancement of medicine, it is sufficient for me to recall to your minds the remarkable transformation which this science has undergone during the course of the last twenty years in a neighboring country of ours—in Germany. Let us for a moment cross over to the other side of the frontier, and in thought go back to about the year 1830. At that time Schelling and his daring "Philosophy of Nature" ruled as absolute lords over the German mind. Poetical notions and transcendental conceptions were then the fashion; and a physician even permitted himself, in a treatise on mucous fevers, to seriously compare a blood-globule with the terrestrial sphere because both were round, flattened at the poles, and both possessed a central nucleus surrounded by an atmosphere.[2]

During that time medicine was reduced to a deplorable condition, although the Germans possessed translations of the principal works relating to pathology, which had just been published in France or England; still the progress accomplished by these last-named countries was for the Germans as though it had never happened, for no one understood the importance of it. Physical diagnosis had never been formulated, either in the hospitals or in private practice.

In more than one German university the stethoscope was almost unknown; when they accidentally ran across one of these instruments, they examined it with a sort of infantile curiosity; or, again, they greeted with sarcastic jests those few eccentric ones who by means of this bit of wood pretended to hear *unheard-of things*. Besides, most of the diseases of the chest and the heart, and chronic affections of the skin, were an almost unexplored region.

Even when they began to take notice of the French, it was only to turn to ridicule, and this time with the appearance of justice, that strange mania which impelled them to consider all diseases as inflammations.[3]

Things remained in this state until about the year 1840. Then the work of regeneration commenced, chiefly through the influence of Schœnlein, by the importation of French methods, and their intervention in the domain of the clinic. Then it became the turn of pathological anatomy, brilliantly represented at Vienna by Rokitansky. But Müller had already appeared with his physiology, and soon he created pathological histology, which was to remain for a long period an almost exclusively German science.

Gentlemen, you know the rest. Then the German universities presented the spectacle, new to them, of an almost unheard-of feverish activity; and you are not ignorant of the fact that that feverish labor, which even now does not show any signs of diminution, has already produced more than one fundamental work.

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[1] "Practice without incessant scientific renovation would very soon become, be assured of it, a belated routine, and one as if stereotyped." Béhier: Leçon d'Ouverture du Cours de Clinique Médicale, p. 19. Paris, 1867.

[2] H. Horn: Darstellung des Schleimfiebers. 2 Auflage.

[3] C. A. Wunderlich: Geschichte der Medicin, p. 332. Stuttgart, 1859.

During more than ten years this grand intellectual movement remained almost unnoticed by the French. From time to time some far-seeing observer endeavored to attract public attention to it, but he had to fight against general indifference; and while all Germany was in activity, we, in France, were occupied with other cares. Finally the day has come, and we comprehend that a grand power has just risen alongside of us, and that it must be reckoned among the sciences beyond the Rhine (*sciences d'outre-Rhin*).[1]

By a very natural reaction we at once exaggerated the tendencies which at first had been combated; and while in France we are to-day much inclined, perhaps, to overvalue German works, our neighbors, intoxicated with success, seem to think that henceforth the realm of science belongs to them alone. We should, gentlemen, pardon somewhat the elation of such a triumph.

But not without regret have we but lately seen an eminent man confound the rights which his high position as a scholar confers upon him, with the political power which was given him by his electors in Berlin, and abuse the word *science* to make the Germans hot-headed at the expense of a strict patriotism.[2]

No one should forget that *science* belongs to no country—belongs, as such, to no race. With the exclusive and illiberal ideas of the Prussian scholar let us contrast the noble words of one of the greatest of England's physicians.

"Reason," says Graves, "Reason has extended its empire from the old to the new continent—from Europe to the antipodes; to-day she has the whole world for her domain, and the sun never sets upon her possessions. Individuals take rest, but the general intelligence of mankind is forever sleepless."[3]

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[1] This lecture was delivered in 1867.

[2] These are allusions to a discourse delivered by Professor Virchow, in Hanover, at the Congress of German Naturalists, September 20, 1865. See the Revue des Cours Scientifiques, 1856–1866.

[3] Leçons de la Clinique Médicale, translated by Dr. Jaccoud. Vol. i., p. 53. 1863.

LECTURE I.

GENERAL CHARACTERISTICS OF SENILE PATHOLOGY.

Summary—Object of these Lectures—Organization of the Salpêtrière Hospital from a Medical Point of View—Chronic Affections; Diseases of Old Age—History of Senile Pathology—Physiology of Old Age—Anatomical Changes in the Organs and Tissues—A complete *Résumé* under the Term *Atrophy*—Exceptions in the Case of the Heart and Kidneys—The Various Derangements Resulting from these Modifications in Structure—Certain Functions Diminished in Old Age, and Others Preserved—Pathological Immunities of Old Age; the Peculiar Impress it gives to the Greater Number of Diseases.

GENTLEMEN:—The lectures which you are going to attend are meant to bring before you in review the most interesting clinical facts which the Salpêtrière Hospital can present to your observation. Those among you, gentlemen, who until now have frequented only the ordinary hospitals, may expect to see pathological cases here exhibited with quite a strongly marked local coloring.

You are, no doubt, acquainted with the internal organization of this vast establishment.[1]

Leaving out of consideration the *employés*, and also the number of lunatics, idiots, and epileptics, who form a separate and distinct class, the remainder of the population of this asylum consists of about twenty-five hundred females, who, with some few exceptions, belong to the least-favored portion of society.

From the standpoint of a medical clinic, which alone should engage our attention, they form two very distinct categories.

The first is composed of women who are, in general, over seventy years of age—for the administrative statutes have so decided it—but who, in all respects, enjoy an habitual good health, although misery or desertion has put them under the protection of public aid. Here, gentlemen, is where we shall find the materials which will serve us in making a clinical history of the affections of the senile period of life.

The second category comprises women of every age—smitten, for the most part, with chronic, and, by repute, incurable diseases, which have reduced them to a condition of permanent infirmity.

In this respect we here possess advantages which those in the ordinary hospitals are, for the most part, deprived of; and we are also placed in the most favorable position for studying with benefit those diseases whose evolution is a slow one.

Indeed, the numerous population of these wards allows us to observe,

[1] Those of our readers who desire more ample details upon the interior arrangements of the Salpêtrière, as well as the history of the institution, can consult with benefit the interesting chapter reserved for The Hospital for the Aged (women), by M. Husson, late Director-General of Public Assistance, in his Étude sur les Hôpitaux. Paris, 1862.

under the most diverse aspects, the principal types of one and the same morbid species; but, what is still of greater importance, we are here permitted to follow the patients through a long period of their existence, instead of being present only at a single episode of their history. Thus we see developed to its utmost limits the pathological process whose initial phase is usually the only one known; in fact, we are called upon to state the organic lesions which characterize the disease when it has been terminated by death.

In other cases—unfortunately but too rare—we see cures effected sometimes spontaneously, sometimes induced by the happy intervention of the art. But that which we learn to recognize here, better than anywhere else, is the value to be attached to those means which alleviate when it is impossible for us to cure.

To-day I purpose calling your attention more especially to the most general characteristics of diseases which supervene upon the last period of life.

I.—The importance of a special study of the diseases of old age cannot be contested at this day. We have come to recognize in reality that, if the pathology of childhood countenances clinical considerations of a special kind, and if it is indispensable that it should be known from a practical point of view, then also senile pathology presents its difficulties, which cannot be surmounted except by long experience and a profound acquaintance with its peculiar characteristics.

And yet, gentlemen, this very interesting portion of medicine has been neglected for a long time, and has only succeeded in obtaining its autonomy in our day.

It was at an epoch very near ours, in France and in this very hospital, that the pathology of old age was constructed and asserted, if I may be allowed the expression, in all its originality. Before that time one could scarcely cite a work in which the slightest indication could be found of the particular physiognomy of the diseases of old age. If you except the little treatise of Floyer, published in 1724, the more recent work of Welsted, and lastly that of Fisher, which dates as far back as 1766,[1] most of the medical works of the past century which touch, in a special manner, upon the senile period of life, have a literary or a philosophical bearing; they are more or less ingenious paraphrases of the famous treatise "De Senectute" of the Roman orator.

It was reserved for Pinel to point out, if not to fill up the gap; however, at the epoch when he wrote his "Treatise on Clinical Medicine," the Salpêtrière Hospital was already organized as it is to-day; the penitentiary which had formerly been part of the institution had been done away with; the infirmary had been founded, and sick women were no longer carried to the *Hôtel-Dieu* at the risk of dying on the way.[2]

But Pinel could not think of restraining his studies to a limited section of science. Much more ambitious views had taken possession of his mind; his idea was to embrace pathology in its vast entirety, and to create a philosophical nosology by applying to medicine the analytical method, according to the rather emphatic language of the eighteenth century. Thus, the differences which separate senile from ordinary pathology are very rarely found described in his writings, although he passed the much greater portion of his medical life in hospitals reserved for the aged.

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[1] Floyer: Medicina Gerocomica. Londini, 1724. Fisher: Tractatus de senio. 1766.
[2] Pinel: Traité de Méd. Clinique. Introduction, p. xiii. Paris, 1815.

To Landré-Beauvais, one of the scholars and successors to Pinel at the Salpêtrière, we owe the first special description which had ever been given of a disease met with at every step in the wards of this hospital, although it is not exclusively a disease of old age.[1]

I speak of nodular rheumatism (*rheumatisme noueux*), called also *arthritis pauperum*, which is a very frequent disease among the indigent classes. Landré-Beauvais designates it by the name of *primitive asthenic gout*, while at the same time he recognizes that it differs from true gout. Here, gentlemen, is a formidable affection on account of the infirmities which it involves; in every respect it deserves to be put in the first rank of those chronic diseases which prevail in this hospital, and to which, later, we are to devote our attention.

The clinical lectures delivered at the Salpêtrière by Rostan, about the year 1830, had immense publicity at that time. Several questions relating to senile pathology underwent a profound study at the hands of that eminent professor.[2]

Two of his works, above all, remain justly celebrated. The first has for its object to prove that the asthma of the aged is not a nervous disease, but one of the symptoms of an organic lesion; and to-day we know that, although this proposition when taken in its general sense is too absolute, yet it is not the less true in the great majority of cases. The second is a remarkable study upon cerebral softening, which has completely transformed all our notions upon the subject. It is known, according to Rostan, that this alteration, which is so frequent at an advanced period of life, far from being the result of inflammatory action, is a senile destruction presenting the most striking analogy to the gangrene occurring in old age; and the researches of observers, aided by all the modern means of investigation, have abundantly confirmed this idea.

The immense amount of material laboriously gathered by Professor Cruveilhier, during his stay in the Salpêtrière, has contributed in a great measure to the construction of an imperishable monument—I mean the "Atlas of Pathological Anatomy." Innumerable observations, which have shed a new light not only upon the pathology of old age, but also upon the history of many a chronic affection, are found accumulated in this vast work.

On account of a more or less direct impulse from the great teachers whom we have just mentioned, several important monographs relating to the diseases of old age have been published by observers who drew the materials for their works from this hospital. I shall only mention the remarkable memoir of Hourmann and Dechambre upon pneumonia in the aged,[3] and the treatise on cerebral softening, by Durand-Fardel.

But, to constitute in reality a senile pathology, these scattered fragments must be brought together in a systematic manner. And such was the end which Prus sought to attain in his "Researches upon the Diseases of Old Age," presented to the Academy of Medicine in 1840. But, in this direction, the French writer had been preceded by Canstatt, in Germany; to the latter author we owe the first dogmatic treatise that has appeared upon the diseases of old age.[4]

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[1] Landré-Beauvais: Thèse de Doctorat, an VIII.

[2] Mémoire sur cette question: L'Asthme des vieillards, est-il une affection nerveuse 1817. Recherches sur un Maladie encore peu connue, qui a reçule nom de Ramollissement du Cerveau. By L. Rostan, Physician to the Salpêtrière. 1820.

[3] Archives de Médecine. 1835–36.

[4] Die Krankheiten des höheren Alters, etc. Erlangen, 1839.

Unfortunately, this work, which bears the date of 1839, was composed under the influence of Schelling's doctrine, which so long reigned across the Rhine, and which bears the ambitious title of the "Philosophy of Nature." In it imagination occupies an enormous amount of room at the expense of positive and impartial observation. Still, we do find in the work of Canstatt ingenious and often true ideas, which insure him an honorable place in science.

An entirely different method inspired the studies of Beau[1] and of Gillette[2] upon the diseases of old age, as well as Durand-Fardel's "Traité Clinique," published in 1854. In addition to these synthetical works, we might cite numerous monographs relating to special points on senile pathology; but we do not pretend to name all, and, besides, we shall have more than one occasion to allude to these works in the course of our lectures.

We shall finish this brief historical sketch by mentioning three interesting foreign works, recently published, from which we shall frequently borrow. The first one is the voluminous "Clinical Treatise on the Diseases of Old Age," from the pen of Dr. Geist, Physician to the Hospital of the Saint Esprit, in Nuremberg. The second is a "Collection of Clinical Observations," written by Dr. Mettenheimer, of the Hospital for the Aged, in Frankfort. The third is the work of Dr. Day, published in London in 1849.[3]

II.—There is a common characteristic which is found in all the writings I have just named to you: this is a manifest tendency to ascribe, as far as possible, the particular points which are distinctive of the diseases of senility to the anatomical or physiological modifications which occur in the organism solely on account of age. We cannot wonder at this, if we notice that nearly all these writings are of recent date, and belong, at least on one side, to the organic school. For the rest, the preliminary study of the modifications under discussion ought, it seems to me, to cast the strongest possible light upon the history of diseases of old age.

We shall have to notice, among other things, that the textural changes which old age induces in the organism sometimes attain such a point that the physiological and the pathological states seem to mingle by an imperceptible transition, and to be no longer sharply distinguishable.

I shall, then, undertake to give a rapid sketch of the anatomy and physiology of the senile condition, but not without remembering that we are always to look upon them from a special standpoint. I shall limit myself to the indication of the most general characteristics, and, when entering upon details, it will be only for the purpose of deducing direct applications to practical medicine from them.

Certain general modifications at once arrest our attention. You are all familiar with the external appearance of an old man—that dry and wrinkled skin, those thin and gray locks, the toothless mouth, the stooping form which is bent upon itself—all these changes correspond to a general atrophy

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[1] Études Cliniques sur les Maladies des Vieillards: Journal de Médecine de Beau. 1843.

[2] Art. Vieillesse, in the Supplément au Dictionnaire des Dictionnaires de Médecine. Paris, 1851.

[3] Geist: Klinik der Greisenkrankheiten. Erlangen, 1860. Mettenheimer: Beiträge zur Lehre der Greisenkrankheiten.

of the individual, for, at the same time the stature diminishes, the weight of the body becomes less, as Quételet has proved.[1]

A more or less pronounced emaciation usually corresponds to these various phenomena. However, you may meet with a different state of being—that is, as they used to say, the *habitus corporis laxus*, characterized by an accumulation of fat beneath the integument and deep within the splanchnic cavities. But this is generally a transitory condition, and it is not long before it gives way to the *habitus corporis strictus*, which is the almost exclusively prevailing state at the period of decrepitude.

That emaciation spoken of is the consequence of a morbid process which exerts its action, not only upon the muscles of organic life and upon the various portions of the skeleton, but also upon the greater number of the splanchnic organs; the brain, the spinal cord, the nerve-trunks, the lungs, the liver—in fact, all the blood-making organs—participate in this retrograde movement; the spleen and the lymphatic ganglia undergo a remarkable diminution in weight and in volume, which diminution advances with age.

But, by a very remarkable kind of contradiction, the physiological reason for which does not yet seem to us to be sufficiently established, the heart and the kidneys are exceptions to this law,[2] and preserve the dimensions found in middle life. Indeed, in many old people the heart is seen to undergo a genuine hypertrophy;[3] this, it seems to me, is a pathological condition dependent upon that arterial degeneration which is called senile. On its side the net-work of capillary blood-vessels grows poorer and poorer, not only in the principal viscera, but also in the deep parts of the skin and the mucous membranes. The latter at the same time lose a portion of their villous and glandular elements in the intestinal canal.[4]

Of what does this atrophic action, which exerts its forces upon the collective organs and tissues, consist? First, and in the highest degree, it is a simple process of atrophy; the cellular elements of the parenchyma, the muscular, and perhaps also the nervous elements, progressively diminish in volume, but without presenting any essential modification of structure; this is pre-eminently noticeable, according to Otto Weber,[5] in the muscles of the aged where the elements are pale, of small dimensions, and all very nearly equal in volume, contrary to what is the case in adult life. Connective-tissue, however, does not participate to the same degree in this work of slow destruction; it is even seen to predominate over the specific elements in the viscera; and this has been well established by Dr. Bastien in the case of the liver and the majority of abdominal organs.

But, in a more advanced stage, atrophy is accompanied by a degenerative action; that is, the elements undergo modifications in their chemical constitution, and become the seat of pigmentary or fatty degeneration and calcareous incrustations. This, for example, is what occurs in the cells

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[1] According to Quételet (Book II., chap. ii.), man attains his maximum weight at about the fortieth year; he commences to lose weight at about the sixtieth, and at eighty he has lost at least six kilogrammes (thirteen and one-fourth pounds). In women, the maximum weight is attained at fifty.—Sur l'Homme et le Développement de ses Facultés. By A. Quételet, Perpetual Secretary to the Royal Academy of Brussels. Paris, 1835.

[2] Rayer: Maladies des Reins. Vol. i., p. 5.

[3] It is hardly necessary to recall here the justly celebrated work of Bizot, inserted in the first volume of the Mémoires de la Société Médicale d'Observation de Paris.

[4] Berres, after Geist, loc. cit. N. Guillot: Recherches sur la Membrane Muqueuse du Canal Intestinal. Jour. l'Expér. Vol. i., p. 161. 1837-38.

[5] Handbuch der allgemeinen und sp. Chirurgie. Vol. i., p. 309. Erlangen, 1865.

of the brain, as we are informed by Professor Vulpian, the perfect exactitude of whose statement I have frequently been able to substantiate.[1] According to Virchow, at the same time that the neuroglia tends to predominate over the nervous elements in the encephalon, it habitually becomes infiltrated by a more or less considerable number of amyloid granulations;[2] the brain-tissue then undergoes a chemical alteration, according to the researches of Bibra, which were confirmed by those of Schlossberger.[3] The fatty materials which enter into its constitution suffer a notable diminution, while, on the contrary, the proportion of water and phosphorus is increased.

And yet, according to Vulpian,[4] fatty granulations are deposited in the primitive muscular fasciculi of animal life, solely because of the progress of age; and this alteration may attain such a point in the lower limbs, where it especially shows itself, that a more or less complete paraplegia is the result. The muscular fibres of organic life do not escape fatty degeneration, and you will frequently have the opportunity to prove that the muscular wall of the heart is almost always the seat of it in women who die at an advanced age. To this alteration in the cardiac tissue can be ascribed the phenomenon of asystolism which is so often observed in old people, even when they seem to be in the best of health.

Indeed, fatty granulations often fill the walls of the cerebral arterioles as Paget[5] has shown, and Professor Robin also.[6] And it has been shown by Vulpian that this senile change is not peculiar to man, but that it is equally encountered in old mammals, in the dog especially.

No one of you, gentlemen, will fail to observe that when these alterations shall have attained quite a pronounced degree, they will go beyond the limits of the physiological state, since they have the power to produce, of themselves, functional derangements which at times are extremely grave. This becomes especially clear concerning that change in the arteries called atheroma, and the calcification which is so frequent an accompaniment of it.

From a standpoint of histological development, atheroma of the arteries tends to separate itself widely from the usual forms of senile atrophy. The latter seems to be the result of a purely passive process; the former, on the other hand, seems, in the first phase of its evolution, to consist in a more or less active proliferation of the elements which are the normal constituents of the internal coat of the artery. At a given time these newly formed elements suffer a fatty degeneration; but this is a consecutive phenomenon. The granulations which have thus been formed accumulate in the deep portions of the internal lining membrane, and it is in these deeper portions where the process first appears and is most strikingly marked: the most superficial layer becomes distended with them, though it still resists for quite a long time. In this way are formed those accumulations rich in fat and cholesterin crystals, which have been designated by the name of atheromatous abscesses. They are sometimes seen to open into the cavity of the artery whose walls they occupy; and their contents, mingled with the blood, may be swept into the general circulatory current, reach ves-

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[1] Leçons de Physiologie Générale et Comparée du Système Nerveux, p. 265. Paris, 1866.

[2] Handbuch der sp. Pathologie. Vol. i., p. 346.

[3] Consult Geist, op. cit., p. 158.

[4] Loc. cit.

[5] On Fatty Degeneration, etc.: London Med. Gaz. 1850.

[6] Mémoires de la Société de Biologie. Vol. i., p. 33. 1850.

sels of a small calibre, and then cause those frequently formidable symptoms of capillary embolism. In a less advanced stage the action of the atheromatous tumor is limited to narrowing, and later on to complete obliteration of the artery which is the seat of the degeneration. Then are produced in various portions of the organism those changes resulting from faulty nutrition, which constitute one of the most original chapters in the pathology of old age. In fact, we see that the majority of the cases of cerebral softening and capillary apoplexy of the encephalon, occurring at an advanced age, result from atheromatous obliteration of the arteries :[1] it is the same with visceral infarctions, with that gangrene of the extremities called senile, and with many other changes as well.

But here we are encroaching upon the domain of pathology, a thing we wish to avoid for the present. I shall now indicate in a few words those physiological modifications which correspond to the textural changes, the summary view of which has just been presented you. If it is true, in a general way, that with the progress of age all the functions are seen to become simultaneously enfeebled, yet it must not be supposed that this proposition is always infallible ; and it is only the analytical study of facts that can afford us any reliable information concerning the true state of affairs.

The generative mechanism and the muscular force in the aged undergo so evident an enfeeblement that it is not necessary to insist upon this point.[2] And with regard to the functions of the nervous system of organic life, it is enough to recall the well-known lines of Lucretius.

> Præterera gigni pariter cum corpore, et una
> Crescere sentimus, pariterque senescere mentem.
> —"De Nat. Rerum," ii., 446.

The functions of the respiratory apparatus, as a whole, are equally weakened, and we find the expression of it in a diminution in the quantity of carbonic acid exhaled, in an augmentation of the number of inspirations, and in the reduction of the vital capacity of the lungs ; this last result, according to the spirometrical researches of Wintrich, Schnepf, and Geist, begins to manifest itself about the thirty-fifth year of life, and reaches its maximum between the sixty-fifth and the seventy-fifth.[3]

Most of the secretions are diminished, the sweat and the urine in particular ; and it is almost beyond question that the senile dyspepsia, upon which our renowned naturalist, Daubenton, has insisted in his great but little-known work, depends in great measure upon a sensible diminution of the gastro-intestinal secretions.[4]

But what are we to think of the functional weakening of the arterial system, when, according to Dr. Marey,[5] the heart of the aged is more powerful than ever, and the arteries present energetic pulsations? It seems proved that, in all cases, the pulse augments its frequency in the senile period of life.[6]

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[1] It is to be clearly understood that we do not speak of intra-encephalic hemorrhages, which have also been rather gratuitously attributed to an atheromatous degeneration of the cerebral arteries. Later there will be an opportunity for further explanation on this point.

[2] Consult Empis : Études sur l'Affaiblissement Musculaire Progressif chez les Vieillards. Arch. de Médecine. 1862.

[3] Geist: op. cit., p. 102.

[4] Mémoire sur les Indigestions, qui commencent à être plus fréquentes chez la plupart des Hommes à l'Âge de 40 à 45 ans. Paris, 1785.

[5] Études sur la circulation, p. 415.

[6] Leuret et Mitivié : Sur la Fréquence du Pouls chez les Aliénés. Paris, 1832. And Geist : op. cit., 85.

We are but slightly acquainted with the degree of intensity which nutrition manifests in the aged; but the use of the thermometer has given us much more precise ideas with regard to calorification. Before having applied this instrument in researches upon this subject, it was believed that the temperature in old age was lower than in adult life; but to-day we know that the heat of the central parts remains about the same in all periods of life. It has even been supposed that the general temperature rises toward the end of life.[1] My own researches tend to prove that the only real difference which exists between aged persons and adults in this regard, is that in the former the axillary is much lower than the rectal temperature, while in the latter there is hardly any perceptible difference between the two.

Here is a woman, one hundred and three years old, and who is in excellent health; the axillary temperature is 37.4° C. (99.3° Fahr.); while in the rectum the temperature is 38° C. (100.4° Fahr.), which is the maximum normal temperature in the adult.

Thus, gentlemen, if old age enfeebles the greater number of our functions, it is far from paralyzing all of them; and rigorous observation shows us that, in certain respects, the organs of the aged perform their tasks with quite as much energy as those of adults.

III.—Gentlemen, the preceding sketch tells us that the progress of age establishes a wide difference in pathological phenomena, by virtue of its physiological modifications.

We shall then study this question from three different standpoints:

First.—There do exist special diseases of old age which, in part at least, arise from the general modifications which the economy has undergone. As examples I shall mention senile marasmus, senile osteomalakia, senile atrophy of the brain, certain alterations of the blood,[2] senile asystolism, and lastly, arterial atheroma, whose study constitutes one of the most interesting phases of medicine in old age.

Second.—Among the diseases which can exist at other periods of life, there are yet several which, during the period of senility, present special characteristics; such, for example, is lobar (*croupous*) pneumonia, that terrible enemy of the aged, and one of the principal causes of mortality in this hospital. We shall later on recur to this part of the question.

Third.—Old age seems to create, in certain regards, pathological immunities. The eruptive fevers, typhoid fever, and phthisis, are quite uncommon at this period; still these immunities must not have their importance exaggerated, for they are far from being absolute, as Rayer has proved in the case of typhoid fever, Murchison in that of typhus, and other authors in cases of other diseases.[3] And who, besides, does not know that Louis XV. died of small-pox at the age of sixty-five?

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[1] Von Barensprung, in Canstatt's Jahresbericht, 1851; and Geist: op. cit., p. 32.

[2] The frequency of intravascular coagulations in the aged seems to prove that there exists in them a tendency to inopexia, and *purpura senilis* comes under this rule; for it is probable that this latter affection arises—at least it does so very often—from the spontaneous rupture of the capillary vessels. (Consult Wagner: Manual of Gen. Path., p. 543, for Inopexia.—L. H. H.)

[3] Consult Rayer: Gaz. Méd., vol. x., p. 573, 1842; and Uhle: Ueber der Typhus abdominalis der alteren Leut., in Archiv für physiol. Heilkunde, Bd. III., Heft. 1859. These authors report striking examples of typhoid fever in the aged. Murchison calls attention to the fact that no age is exempt from typhus: from the fifteenth to the twentieth year the proportion is 16 in 100; from the sixtieth to the sixty-fifth, it is 2.5 in 100; from the seventieth to the seventy-fifth, it is 1.21 in 100. The case

I think enough has been advanced, gentlemen, to convince you that a *senile* pathology does exist. And to offer you a striking example of the modification which age may impress on manifestations of disease, we shall study, at our next lecture, the febrile state in old age, and endeavor to point out the analogies and the differences existing between it and the febrile condition in the adult.

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which occurred at the most advanced age was one in a man of eighty-four. Relapsing fever is less frequent than typhus in old age, although several examples of it have been observed; over fifty years the proportion is 6.63 per 100; over sixty it is 1.6 per 100. Old women are more exposed to these two diseases than are old men. For typhoid fever the proportion is 1.46 per 100 over fifty years; and 0.5 per 100 over sixty. These figures are enough to show that the relative immunity enjoyed by the aged in respect to the continued fevers is far from being absolute.—A Treatise on Continued Fever in Great Britain, pp 61, 303, and 410. London, 1862.

Now, as regards phthisis, Vulpian and myself have noticed that tuberculization is more frequent in the Salpêtrière than is generally supposed. One of Vulpian's students, Mr. Moureton, in his inaugural thesis, reports nine cases of acute tuberculosis in the aged. Three of the patients were over eighty, and acute phthisis was *primary* in all of these cases except one.—Thèse de Paris. 1863.

Each year in the Salpêtrière we notice some cases of cerebro-spinal meningitis foudroyante. Since 1852 I have gathered quite a number of facts of this kind, which will be found collected in the thesis of Dr. Inglessis.—Sur quelques Cas de Méningite Cérébro spinale observés à la Salpêtrière pendant le printemps de 1852. Thèses de Paris. 1855.

LECTURE II.

THE FEBRILE STATE IN THE AGED.

Summary.—Want of Reaction in Old Age—Organs seem to Suffer separately—Latent Diseases—The Gravest Lesions may pass Unnoticed—Fever in the Aged—What is Fever?—Importance of the Clinical Thermometer—Chill in Old People—Temperature-Curves of Lobar Pneumonia—The Practical Deductions to be made thereform—Defervescence, Crises, and Critical Perturbations—Diseases where the Temperature is Lowered instead of Elevated.

GENTLEMEN:—At the last lecture I endeavored to bring out the particular stamp which old age imprints on all morbid manifestations. I then relied principally on the data of physiology; but to-day we are to pursue this study remaining exclusively on clinical territory.

Not only does old age have special immunities and pathological predispositions unknown to the adult, but we also see that, in old age, the general reaction which we commonly meet with in disease has undergone a complete transformation. In this period of life the organs seem, as it were, to become independent of one another; they suffer separately, and the various lesions to which they may become subject are scarcely echoed by the economy as a whole. Thus do the gravest disorders manifest themselves by slightly marked symptoms; they may even pass unnoticed, and it is in old age that we observe the greatest number of latent diseases.[1]

This point of view, gentlemen, is so important in practice that it deserves to be made more conspicuous; I shall therefore give you some appropriate examples of it.

Let us lay aside for a moment the study of physical signs, whose importance is elsewhere so great in every respect; let us forget the difficulties which attend auscultation and percussion in old age; we shall have occasion to recur thereto later on. Let us attend only to those phenomena of sympathetic reaction whose absence, often complete, may certainly be fit cause for wonderment.

Let us take as an example one of the most frequent affections met with in this hospital. I mean biliary gravel, which commonly induces in adults general phenomena of great intensity. You are acquainted with the formidable aspect of hepatic colic, which occurs with such an array of terrible symptoms, which, once seen, are never forgotten. Now, you will learn with surprise that in the aged it is often difficult to recognize its symptoms, so diminished are they; at best we only find a little weight in the right hypochondrium, a few attacks of vomiting, slight jaundice, sometimes delirium

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[1] "In advanced age the organs seem to act and suffer separately—their sphere of activity appears more restricted. . . . One should never forget that in advanced age the gravest lesions may coincide with a small number of slight, almost insignificant symptoms."—Grisolle; Traité de la Pneumonie. First edition, p. 425.

and cerebral symptoms; and these are more apt to induce error in the diagnosis than to enlighten us as to the nature of the disease.[1]

If the biliary passages, when distended by the passage of calculi, show such a very slight disposition to induce a general reaction, it is the same in the case of the renal excretory passages, which may suffer the contact of urinary gravel almost without pain; and, in this way, the intense pains of nephritic colic are very nearly wholly unknown in old age.

In another kind of cases we see diabetes occurring in patients of an advanced age with very different symptoms from those which characterize it in the adult. The urine, often but slightly increased in amount, contains sugar only intermittingly;[2] and thirst, that betraying symptom, which most frequently puts us on the way of the diagnosis, may be wanting completely in the aged with diabetes.

Following such a statement of facts, you will learn without astonishment that cancer of the stomach[3] and of the liver, as well as pulmonary tuberculosis,[4] may remain in a latent condition during the whole course of their development. Such are surprises which the autopsy very often prepares for us.

But in lobar (*croupous*) pneumonia, so frequent in this hospital, the almost complete absence of general signs is, more than anywhere else, most strikingly exemplified. It is enough to quote, in this connection, a passage from the important memoir of Hourmann and Dechambre.[5]

"Old women," say these authors, "do not even complain of malaise; no one in their dormitories—neither among attendants, house-maids, nor neighbors—notices any change in their condition. They get up, make their beds, walk about, eat as usual, and afterward, feeling a little tired, they totter to their beds and expire. That is what in the Salpêtrière is called *sudden death*. The cadaver is opened, and a large portion of the pulmonary parenchyma is found suppurating."

Do not such accounts seem very strange? Is it not, however, an acknowledgment that the laws which, in adult life, govern the relationship between symptoms and lesions, are completely inverted in the aged? No, indeed; there is reason for remarking, at once, that these kinds of facts, though they may not be called in question, yet always ought to be considered as exceptional. And besides, they are not altogether uncommon in ordinary pathology. Pneumonia sometimes remains latent in adults, in

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[1] "In the Salpêtrière infirmary few autopsies are made without there being found a greater or smaller number of calculi in the bladder, yet biliary colics are extremely rare in the Salpêtrière."—Beau: Études sur l'Appareil Spléno-hépatique. Arch. de Méd., p. 401. April, 1851.

". . . . The fact holds good for colic with all its train of painful symptoms; but you must take into account the diminution of sensibility; and it is not rare to find dull pains in the gastro-hepatic region, pains which the patients always ascribe to imaginary causes, but which may certainly have been caused by the presence of calculi."—Gillette: article cited, p. 898.

I wholly agree with Gillette in this respect, and I may add from personal observation that the dull pains may also coincide with the passage of urinary gravel, and this much oftener in the old than in adults.

[2] Bence-Jones on Intermitting Diabetes, and on Diabetes of Old Age: Medico-Chirur. Trans., vol. xxxvi. 1853.

[3] "It is in the aged that we discover those degenerations of the stomach which have a deceptive progress, which are accompanied neither by vomiting, violent pains, nor by dyspepsia—at least the subjects thereof avow not."—Gillette: loc. cit., p. 898.

[4] "Phthisis in the aged is notable on account of its latent and insidious form."—Gillette: loc. cit., p. 898.

[5] Archives de Médecine, vol. xii., p. 57. 1836.

certain particular conditions of the organism, especially in drunkards. One can compare several other grave affections with this same type; for example, who does not know that hemorrhagic small-pox may assume, at the outset, favorable appearances, only to be abruptly contradicted by a fatal termination? But it is, above all, in the class of infectious and contagious fevers that we can affirm facts analogous to these. Thus, in *epidemic yellow fever*, in the *plague*, and in *typhus fever*, there are cases where the great damage to the organism reveals itself by no symptom which in any way predicts the gravity of the disease. Here the pulse is normal, or not far from it; the tongue is clean; the skin is cool, or but slightly warm in the region of the stomach and liver; the mind is cheerful, and the bodily forces unimpaired. But suddenly attacks of black vomiting set in, and death unexpectedly supervenes. An American physician, Dr. Caldwell, to whom we are indebted for a fine treatise on yellow fever, applies the name *walking cases* to those insidious forms in which the unfortunates, believing themselves scarcely unwell, and continuing to carry on their business to the last moment of their existence, have been suddenly struck down by death.[1]

These insidious forms, then, do not exclusively belong to senile pathology; but, if we leave aside these few infrequent cases, and regard only the ordinary clinical ones, we are led to recognize the fact that, as a general rule, there is a want of correlation in old age between the local lesion and the exhibition of general symptoms. A similar state of affairs exists in childhood, as Gillette has ingeniously pointed out to us,[2] but there it is just the reverse. In that period reaction is, as it were, exaggerated and tumultuous, and a violent derangement of the functions is very far from proving a serious danger. In old age, on the contrary, the organism remains impassible, so to speak, in the face of the gravest changes. Reaction is here, then, defective, even to the extent of total absence; and hence, the physician ought to be doubly attentive to, and appreciative of the slightest symptoms, unless he wishes to be surprised by completely unforeseen occurrences.[3]

It is now time to leave this very general standpoint we have just taken, and to broach at last the question which ought specially to claim our attention to-day. We wish to study the febrile state in the aged, comparing it with that of the child and the adult; and, in order to give more precision to the ideas we desire to set forth, we shall choose, as the type therefor, lobar pneumonia—that disease which is febrile in the highest degree, and is common to all ages of life. Its development will enable us to state the deviations which age may induce in one of the chief symptoms of the majority of acute diseases.

"Fever," says Gillette,[4] in a passage where he echoes the opinion of all

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[1] Med. and Phys. Mem., containing a Particular Inquiry into the Origin and Nature of the Late Pestilential Epidemics of the United States. Philadelphia, 1801.

[2] Loc. cit., p. 873.

[3] It must also be remembered that, in old age, sympathetic phenomena sometimes assume an entirely unusual aspect. Thus, pneumonia may assume a masked form, and at one time appear as cerebral apoplexy, with complete resolution and coma, and at another under the guise of a true hemiplegia, with or without contraction of the paralyzed limbs. I particularly emphasize these *pneumonic hemiplegias*, of which Vulpian and myself have met several examples. They always terminate fatally, and we have been able to convince ourselves that there is no corresponding encephalic change. In children, pneumonia may present a cerebral variety characterized by eclampsia or coma.

[4] Loc. cit., p. 874.

the professional writers who had preceded him—"fever in the aged is characterized by an acceleration of pulse and a dryness of the skin, without there being any sensible increase in temperature." He next calls attention to the fact that the initial chill is scarcely noticeable, or is wholly wanting, and that it is the same with the sweatings. The other accessory phenomena of the febrile state are, according to the same author, all more or less extensively modified; and finally, the description which he gives offers a striking contrast to what, in other periods of life, constitute the appurtenances of the febrile condition.

Is it an exact picture, and does it faithfully represent the truth? We must acknowledge that it does not absolutely satisfy us; but, in order to justify our restrictions, it becomes necessary to enter into a preliminary discussion.

What is fever? What is meant by the febrile state?

It is hardly necessary to premise, gentlemen, that the definition we seek is in all respects descriptive, and that we make no pretensions to penetrate the ultimate nature of the phenomena we wish to characterize.

In the days of Hippocrates, at a time when they did not practise the examination of the pulse, elevation of temperature was the one and only element of fever. Galen's definition is sufficient testimony on that point: *calor præter naturam*, this was the characteristic of the febrile state for that great physician.[1] During a long series of ages tradition has respected Galen's opinion; but in time it changed, and we see Boerhaave, under the influence of the iatro-mechanical ideas prevailing in his day, declaring that "acceleration of the pulse is the only symptom which we always find present in fever from beginning to end, and which alone is sufficient for the physician to recognize the presence of fever."[2] Since then the question has been taken up many times, and settled in many and various ways; but it must certainly be acknowledged that to-day the unanimous testimony of modern study has pronounced in favor of the opinion accepted by antiquity. On all sides it has been recognized and proclaimed that a rise in animal heat is certainly the fundamental event of the febrile state. Among the other phenomena that accompany it there is none, not even the acceleration of the pulse, which appears in such a constant, such an obligatory manner. Fever does not exist when the temperature remains at the normal point, and the pulse may attain the limits of utmost frequency, and still no fever may be present. It is sufficient to mention the extreme and excessive excitement of the circulatory system noticed in certain cases of arterial pulsation, and particularly in the exophthalmic and hysterical cachexiæ.[3] Now, can it be said, on the other hand, that fever occurs every time the temperature rises? That is a point on which it is scarcely possible to give an opinion to-day. We really observe, however, an elevation in bodily heat in those cases which seem foreign to all pyretic reaction: in tetanus, in attacks of epilepsy, and in cholera especially at the moment of death; the temperature may then reach 107½° Fahr. or 109½° Fahr. But there is, no doubt, some element which escapes us; it is always the increase in animal heat which predominates in fever, and it may, in many instances, serve to measure its intensity.

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[1] De different. febrium, cap. i. De generali febrium divisione. There is another definition of Galen's, in which the frequency of the pulse is adduced; but it occurs in a less authentic work.

[2] Aphorism 570.

[3] Briquet: Traité de l'Hystérie, p. 326.

It is the methodical use of a means of investigation unknown to the ancients which has, in great measure, contributed to definitely fix our ideas on this question—I refer to the clinical thermometer. Though criticism has not spared it, this means of investigation has made its way in our times, and we can foresee that the period is not far distant when its general use shall have spread throughout the ordinary clinic.

It is said that the celebrated Swammerdam, of Holland, was the first, in the seventeenth century, to think of appreciating by means of the thermometer the heat of disease.[1] Since that time several physicians have engaged in similar methods of observation. In 1754 de Haen called the attention of his students to the necessity of substituting the use of the thermometer for the application of the hand in determining the bodily temperature. And to him also we owe the establishing of an important fact—to which we shall very often allude, because it is so frequently observed in the clinic in old men—that, at the very moment when the skin of the fever-patient is pallid, purple, and cold (*pâle, violacée et refroidie*), in consequence of the contraction of the superficial capillary vessels, the temperature of the blood rises several degrees higher than the normal standard; and that this is not a transient rise, such as occurs in the initial chill of fever, but is, as it appears, a permanent one during the whole duration of the febrile state.

In the past century John Hunter was almost the only author who responded to this call. But in our time the labors of Professor Gavarret (1839), of Bouillard, Monneret, and some other French physicians, have enabled us to appreciate all the clinical import of this means of investigation. Still it is only during the last few years, and in Germany, that any actual progress has been realized in this respect. It may be asserted without any exaggeration, that, in the hands of Bärensprung, Traube, Michaël, and Wunderlich pre-eminently, clinical thermometry has undergone a radical transformation. The question is no longer, indeed, to ascertain that the temperature has risen a few degrees in fever, nor to note its intensity according to the morbid species. The phenomenon must be followed day by day—hour by hour, so to speak—from its very commencement to its definite termination, and in all the varied phases of its evolution, in order to take cognizance of its slightest oscillations, and to show that the graphic tracings obtained by this methodical investigation give constant types for every form of disease, with variations which correspond to the most important circumstances attending the malady. For this is the only way by which one can prove that these tracings have a real clinical importance, or that they enable us to follow the course of the morbid process better, perhaps, than any other method does, and to recognize its various appearances, thereby, as a consequence, unquestionably furnishing valuable information pertaining as much to the diagnosis as to the prognosis. Finally, it must be shown that the thermometrical curves are modified according to certain rules and certain laws, either as the disease has been left to itself, or has been treated by the methodical exhibition of this or that medicinal agent; for we may be allowed to hope that therapeutical experiments may find a criterion of almost mathematical precision in the application of this method.

And this is the complex task imposed upon themselves by the authors we have just mentioned; and though they may not have attained the end proposed, it would be rank injustice not to acknowledge that they have at least disseminated many truths along their route.

The need of similar work seems at last to be understood in France as

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[1] Requin, vol. i., p. 91.

well as in England, and many physicians of both these countries are engaged in this line of investigation.[1]

For nearly three years we have endeavored, as often as has been possible, to make those clinical observations we have been speaking of, upon the aged patients placed under our charge in this hospital; for up to that time the practice of thermometry had generally been confined to children and adults. The results obtained will enable me to present you with a few brief considerations upon the modification which temperature undergoes, in the senile period of life, during the different phases of development of the febrile state, and thus to compare this time of life with the other epochs.

But I am most anxious to have you observe all the advantages which accrue from thermometry in the clinic of the aged. It is hardly necessary to repeat here that our descriptions will apply chiefly to the fever that accompanies lobar pneumonia, though several allusions will be made to other forms of the febrile state.

I.—Old people seldom have a chill, says Beau,[2] and we have seen that Gillette gives very nearly the same opinion. This proposition is much too absolute; for more than once have we witnessed violent and prolonged chills in the aged at the commencement of pneumonia, erysipelas, or synocha[3] (*continued fever*), a very common disease at the Salpêtrière at certain times of the year. These chills, characterized by convulsive trembling, cyanosis, and algidity of the extremities, appear with even a still greater intensity in the paroxysms of symptomatic intermittent fever, which so often accompanies deep-seated suppuration, attacks of visceral phlebitis, and those inflammations of the biliary passages which in old age are so readily induced by the presence of hepatic calculi.[4]

And still, in the midst of all these phenomena, at the very moment when the external surface of the body preserves all the marks of considerable coldness, the central heat is maintained at a very elevated degree. The axillary temperature, it is true, does not enable us to discover all the intensity of this reaction,[5] but in the rectum the temperature runs up to 104° or 105.8° Fahr., as I myself have noticed on many occasions.

This rapid rise in temperature in the beginning of diseases very closely corresponds to what is observed in the case of adults, and, in this one respect at least, old age yields nothing to youth. But this rapidity of inva-

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[1] Consult, in this connection, the excellent theses of Messrs. Maurice (Paris, 1855), Spielman (Strasburg, 1856), Hardy (Paris, 1859), and Duclos (Paris, 1864). In England, Sidney Ringer has made important investigations, and these will be found described in Aitken's work.

[2] Études Cliniques sur les Maladies des Vieillards: Journal de Beau, p. 292. 1843.

[3] Relapsing fever, according to Tanner and Reynolds.—L. H. H.

[4] If hepatic colics are rare in advanced life, it is, on the other hand, very common to see *suppuration of the biliary passages* caused by calculi, and above all by intra-hepatic gravel. This lesion evinces itself externally by an *intermittent symptomatic fever*, in which the beginning of each paroxysm is marked by an intense chill; in the intervals the thermometer often shows the existence of complete apyrexia. Death almost always results from such an occurrence as this. Cornil, in the Mémoires de la Société de Biologie (1865), has published several cases of this kind which were gathered in my service. It is well known that Monneret had already described the existence of a fever of the remittent or intermittent type occurring in diseases of the liver.—Archives de Méd., 1861.

[5] In such cases there almost always exists a difference of a fraction of a degree, sometimes even a whole degree, between the axillary and the rectal temperature.

sion is met with only in certain diseases; there are others where the febrile temperature is reached slowly and gradually; and to remain within the confines of senile pathology, we shall quote as examples, broncho-pneumonia and mucous fever.[1] Besides, it is rare to find the temperature rising to the same degree in these last-named diseases as in lobar pneumonia (*croupous p.*), a fact we shall have frequent occasion to verify. And now, gentlemen, let us see what are the characteristics of general reaction in that phlegmasia which we have chosen for the type of febrile affections of old age.

II.—A chill is the initial symptom of the disease, which from that moment undergoes a regular development. This is the time when it becomes interesting to watch, with scrupulous attention, the daily progress, and, by means of the thermometer, to ascertain the smallest variations in the animal heat; for, in the majority of cases, they correspond with the greatest exactitude to the various phases of the affection.

A momentary improvement habitually follows the initiatory chill; the temperature sometimes falls more than a degree and a half, and the patient experiences a comparative amount of comfort. But this is a deceptive lull, and that very evening, or the next morning, the disease proceeds on its course. The temperature rises again and reaches 104° Fahr.; and when it keeps at this point for several days, you are justified in believing you have a severe case to deal with; but when, on the other hand, it tends to fall progressively to 102° Fahr., or even a little below this, the prognosis is, relatively speaking, favorable. The figures I have just given you correspond to the *evening* temperature, for fever in pneumonia (even lobar pneumonia), does not follow a continual, sharply defined course; there are daily remissions which, in the morning, show a thermometrical difference of nine-tenths of a degree Fahr., on an average. But in catarrhal pneumonia these variations are very much more strongly marked; these are shown by runs of a degree and a half Fahr., two degrees and a half Fahr., and even more than this in some instances. Now, if you remember at the same time that in this latter disease (*lobular p.*) the temperature rises slowly, by successive steps, and scarcely ever reaches the height which we notice in lobar pneumonia, you will readily comprehend that an inspection of the thermometric tracings may often, alone, enable us to distinguish between these two diseases, whose differential diagnosis is at times quite difficult.

We have submitted for your inspection a few examples of this kind, several of which were taken in our wards. A simple glance at them is enough to grasp the differences we have been endeavoring to make manifest.

The juxtaposition of these temperature-curves will enable you to compare at the same time the tracings obtained from the child and the adult, in either the croupous or the catarrhal form of pneumonia, with those taken from an aged patient. A rapid glance suffices to show the perfect analogy between them.[2]

Among the tracings which we have before us, there are two which are intended to bring out in relief the influence exerted upon the variations in

[1] Catarrhal or mucous fever; sometimes synonymous with catarrhal pneumonia. —L. H. H.

[2] Touching pneumonia in children, we have borrowed the thermometrical tracings from the work of Hugo Ziemssen—Pneumonie des Kindesalter. Berlin, 1862.

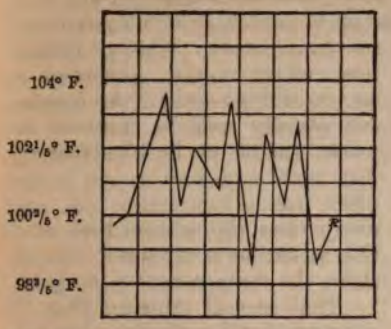

FIG. 1.—Catarrhal pneumonia in a child. Recovery. (Ziemssen.)

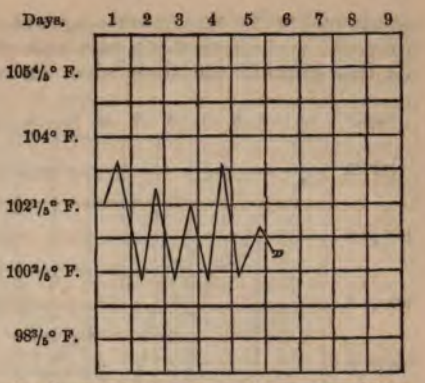

FIG. 2.—Catarrhal pneumonia in a woman eighty-three years old. Death. (Charcot.)

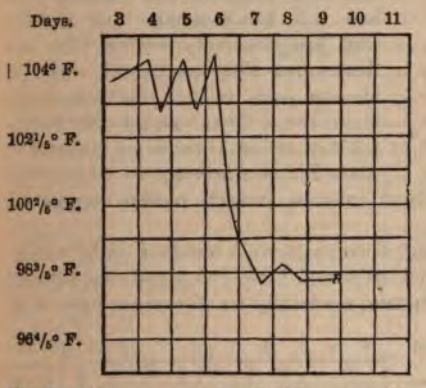

FIG. 3.—Lobar pneumonia in a child three years old. Recovery. (Ziemssen.)

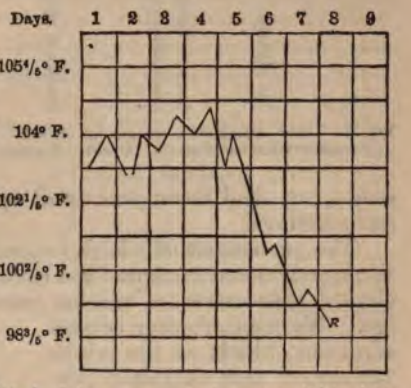

FIG. 4.—Lobar pneumonia in a man thirty-eight years old. Recovery. (Wunderlich, quoted by Aitken.)

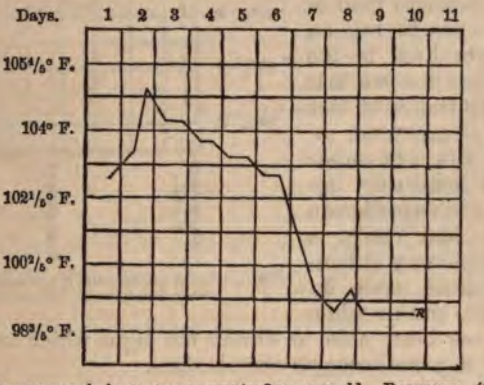

FIG. 5.—Lobar pneumonia in a woman seventy-five years old. Recovery. (Charcot.[1])

[1] In the temperature curve bearing Centigrade degrees, 39° has been, I think, accidentally omitted, and 38° written instead. I have ventured to make this correction.—L. H. H.

temperature by therapeutic agents. In the first of these two drawings (Fig. 6), you see that the administration of large doses of digitalis resulted, on two different occasions when an exacerbation occurred, in a marked fall in temperature. This phenomenon, which never *spontaneously* occurs at this period of the disease, can equally well be induced by tartar emetic or by bloodletting. But such a defervescence is too often but a temporary one, and every time the administration of the medicine is arrested, you observe the temperature rise again.

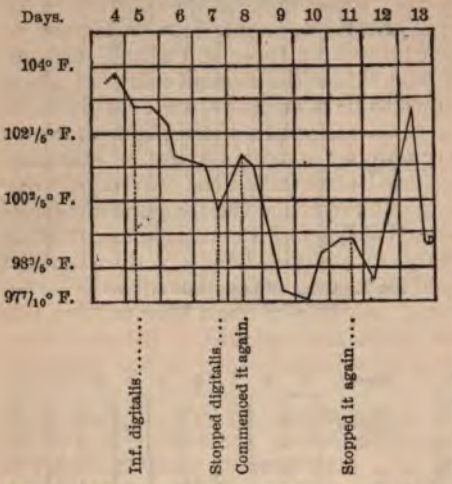

FIG. 6.—Lobar pneumonia, treated by digitalis, in a woman seventy-one years old. Death. (Charcot.)

The second drawing (Fig. 7) shows the effect of administering rum in draughts of 120 grammes to the dose (3¾ ounces). During the first stage of the disease you see the temperature fall slowly but progressively under the influence of this treatment, only to rise as soon as it is discontinued; the disease then assumes its natural career and ends in recovery. But this is a subject to which, no doubt, we shall recur, and dwell upon at greater length in the course of our meetings.

The persistence of a high temperature without well-marked daily variations, and its continuance for a certain number of days, constitute important clinical characteristics of lobar pneumonia, *especially in the senile period of life*. We could mention several affections, which, in the adult, partake of this character (the eruptive fevers and exanthematous typhus, for example). But in old people we shall rarely find it elsewhere than in erysipelas, if we are to rely on what we observe here in the Salpêtrière. Thus you see that the clinical importance of this fact cannot be exaggerated, especially when you remember the difficulties habitually opposed in old age to auscultation of the chest. And really, a diagnosis of pulmonary inflammation is oftentimes made by the thermometer, quite a while before the ear has been able to obtain the signs which evidence this disease, by means of a stethoscopic examination.

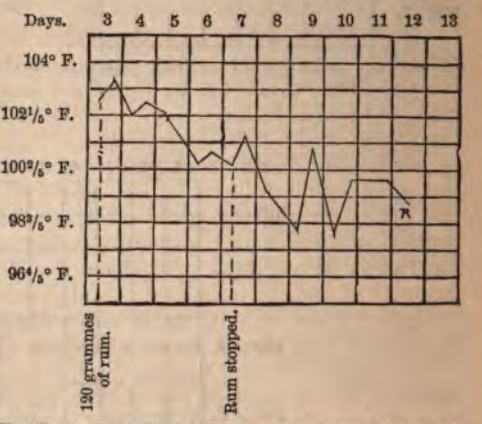

FIG. 7.—Lobar pneumonia in a woman seventy-six years old, treated with rum. Recovery. (Charcot.)

III.—It remains for us to describe the minute details which a thermometrical investigation affords in the period of decline of the febrile state in

the aged, or, as they say in Germany, during the *defervescence*. Sometimes the fall in temperature which marks the return of the normal state is effected by successive steps during a period of three or four days, as occurs in mucous fever or broncho-pneumonia ; sometimes, on the contrary, it occurs very rapidly, and in the course of twelve, twenty-four, thirty-six, or forty-eight hours, the temperature falls two or three and a half degrees, Fahr., or even more than this. At least this is the natural course of affairs in lobar pneumonia, when the issue is to be a favorable one. This rapid fall of the fever is often preceded by a rapid elevation of temperature, which is accompanied by a more or less pronounced and often alarming exacerbation of the symptoms. This is what used to be called the *critical perturbation*. We shall, finally, notice that in defervescence the temperature sometimes falls below the normal standard, remains so for several hours, in some cases during an entire day, afterward to assume definitely the standard which corresponds to the state of perfect health. They have then gone beyond the mark, if one may say so ; but only in rare cases have we seen this fall of temperature below the normal limits accompanied, in the case of the aged, by more or less alarming signs of collapse, which in less advanced years quite often distinguish it.

In many subjects defervescence is the signal for critical phenomena which are to decide the disease ; these phenomena occur either just at the moment when the temperature falls, or a little later on ; but the latter is more *frequently* the case. (See Fig. 10.[1])

In this respect there is no real difference between old people and adults ; except that the sweating-crises, which are so frequent in the middle period of life, are not very often observed in old age, whereas critical diarrhœa, on the other hand, is quite a common occurrence.

You can follow the thermometrical tracings (Figs. 1 to 7), which show defervescence in the child, the adult, and the aged, and recognize very readily that its phenomena are subject to identical laws in all periods of life.

Up to this point we have only taken into consideration those cases which terminate in recovery ; but when the disease is to take a fatal turn, the temperature, which up to this time has maintained the ordinary limits, rises suddenly, within a day, or only within a few hours, one and a half or even three and a half degrees Fahr. higher. Under these circumstances, in adult life, death usually supervenes in cases of lobar pneumonia, and the same result follows in old age in the vast majority of cases. But in the latter we observe, quite often, a mode of termination which is the exception in middle life : instead of rising, the temperature progressively falls for two or three days, until it reaches 100.4°, or even 99.5° Fahr., at the moment of fatal termination. And defervescence to this ominous standard is observed not only in those cases where the patient has been counter-stimulated, but also in those where the disease has been left to itself.

The four following figures (8, 9, 10 and 11) exhibit the thermometrical tracings of two cases of pneumonia which terminated fatally with an elevation of temperature, and one case in which recovery followed.

The fourth (Fig. 11) is a case where death occurred in the defervescence. All the tracings were made here in the Salpêtrière.

Gentlemen, I have shown you, I believe, all the importance of clinical

[1] Consult, in this connection, [the work of Traube: Ueber Krisen und kritische Tage. Berlin, 1852.

thermometry in the study of diseases of old age, and the utility of the results to which it leads, from the triple standpoint of diagnosis, prognosis, and treatment. But what I am most anxious to prove to you is that febrile phenomena, observed at the most widely separated periods of life,

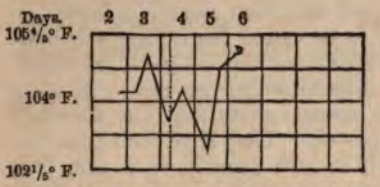

FIG. 8.—Lobar pneumonia in a woman eighty-three years old. Death, with elevation of temperature. (Charcot.)

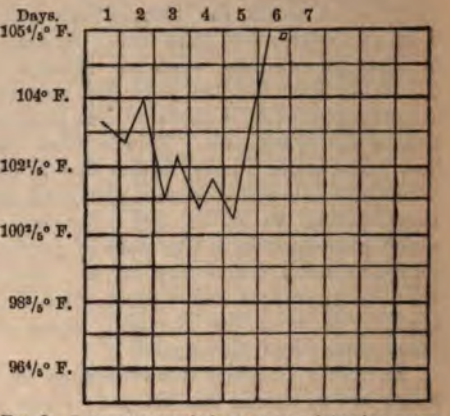

FIG. 9.—Lobar pneumonia in a woman sixty-nine years old. Death, with elevation of temperature. (Charcot.)

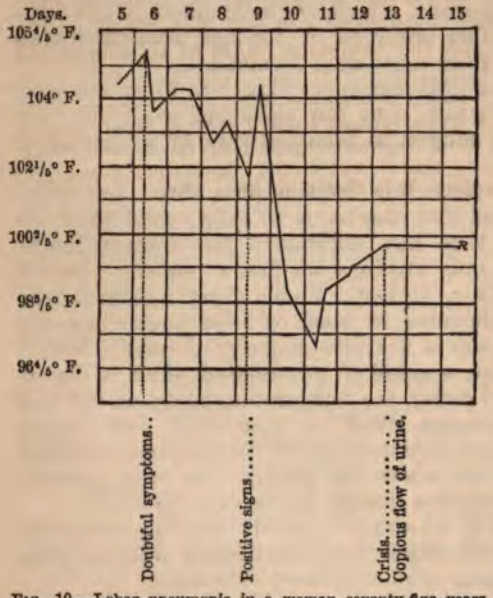

FIG. 10.—Lobar pneumonia in a woman seventy-five years old. Recovery. (Charcot.)

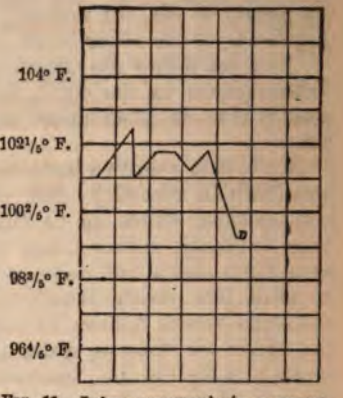

FIG. 11.—Lobar pneumonia in a woman seventy-five years old. Death in the defervescence. (Charcot.)

are at bottom the same, and act in obedience to laws which are identical. Just as in the adult, so general reaction exists in the aged; but it is latent, and its manifestations must be sought for in the central regions of the economy.

Here, then, it becomes very important to distinguish between the results obtained from an axillary and from a rectal thermometrical investigation. In regard to temperature, the axilla corresponds to the surface of the body, and the rectum to the internal viscera. It is true that, in the majority of cases, the curves which correspond to the temperature of these two parts are very nearly parallel—in the adult we may almost say that they coincide; but, in old age, that which corresponds to the axillary temperature is a little lower than that of the rectum. But there are cases, and these are most grave, where wide deviations occur. The external temperature falls, while that of the central regions of the body rises, and the difference between them may be several degrees. Now, in such cases, it is evident how very inadequate the results of axillary thermometry would prove, and how little we could rely on them *exclusively*.

Hitherto, gentlemen, I have scarcely spoken of any diseases, excepting those in which the temperature rises *above* the normal standard. But there is a large number of diseases, especially in the period of old age, which give rise to inverse phenomena, by inducing an actual lowering of the temperature of the central portions of the economy. Without considering cholera,[1] whose effects in this regard are universally known, we may adduce, as examples, certain diseases of the heart, pericarditis, senile gangrene, marasmus, and the various forms of cancer. This algid condition is a very grave symptom, and one which demands prompt relief. And this algidity cannot be revealed except by means of the thermometer, which, consequently, is here called upon to render a new service.

But to-day I do not wish to broach this subject, for no doubt I shall have occasion during our meetings to present you with the result of the researches I have undertaken upon the semeiotic value of algidity in old age.[2]

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[1] During the last cholera epidemic which ravaged Paris, I had the opportunity to prove that the temperature of the central regions remained at the normal standard during the course of the disease, and that it rose on the approach of death, precisely as Doyère had already stated.—Mém. de la Société de Biol., 1866.

[2] See Appendix, Lecture XX.

LECTURE III.

NODULAR RHEUMATISM (*Rheumatisme Noueux*) AND GOUT, PATHOLOGICAL BLOOD-CONDITIONS OF GOUT.

Summary.—Frequency of Chronic Articular Rheumatism in the Salpêtrière—Its Resemblance to Gout—The Doctrine of Identity—Silence of the Physicians of Antiquity in this Regard—Necessity of taking up the Preliminary Study of Gout before that of Chronic Rheumatism—The Gouty Diathesis—Its General Characteristics—Regular and Irregular Gout—Acute and Chronic Gout—Pathological Blood-conditions of this Affection—Gouty Concretions composed of Urate of Soda—Uric Acid's Normal Existence in the Blood—An Excess of it in the Blood of the Gouty—The "Thread" Process—Uric Acid not in Excess in the Blood of Rheumatic Individuals—Gout not the only Disease which coincides with this Alteration—Accessory Changes in the Composition of the Blood in Gout—State of the Urine in Acute Gout during the Attacks and the Intervals between them; in Chronic Gout.

Gentlemen:—In the two preceding lectures we have studied the general characteristics which diseases present during the senile period of life; to-day I purpose to invite your attention to one of those chronic affections which is most commonly encountered in this hospital.

I refer to *chronic articular rheumatism*, which most certainly constitutes one of the most common infirmities of females, at least those of the poorer classes; indeed, we find it in the proportion of eight in one hundred among the infirm women in the Salpêtrière.

It seems to me, then, that from a practical point of view, this question deserves to have your most careful attention, and all the more so since the pathological history of the disease presents many difficulties to be unravelled, many points to be cleared up. For quite a long period of time this apparently unpromising study has been neglected; and notwithstanding the importance of several recent works, there yet remains a wide field for future progress in this direction. Now, only those observers who are in such a hospital as the Salpêtrière can undertake a work like this; but, in order to realize this expectation, it is absolutely indispensable that observations should be made of a very large number of patients, so that a more reliable comparison may be made between the types of chronic articular rheumatism, which are often so diverse in character.

But a difficulty meets us at the very threshold of the question. If rheumatism and gout, when regarded in a general manner, coincide in many respects, and seem to offer in very many ways a profound analogy, it must be acknowledged that it is especially in the chronic form of these two diseases that the resemblance becomes striking, and then may confuse the observer.

We are, therefore, almost, in spite of ourselves, led to trench upon a doctrinal question, and ask ourselves whether, in a general description, we are to confound these two affections, or to draw a radical distinction between them.

We know that the great physicians of antiquity pronounced themselves in favor of the first of these two opinions; or, to speak more exactly, they seem never to have suspected that there was any problem needing their solution. Under the name *arthritis*, or articular disease (*articulorum passio*), they have left us a description of an affection in which we discover at one time the characteristics of gout, and at another those of rheumatism; and this ancient tradition was maintained during centuries, until the time when Baillou mollified the primitive acceptation of the word *rheumatism* and applied the term to that group of symptoms which we know to-day under the name of *acute articular rheumatism*. At a little later period chronic articular rheumatism, in its turn, acquired an autonomy which had long been contended for.

But the distinction which Baillou made, and which never obtained unanimous assent, is to-day sharply combated by eminent investigators; and although most physicians recognize a profound difference between gout and rheumatism, the doctrine of identity has yet found defenders among our contemporaries whose names are authorities in science.

This assent, gentlemen, is in great part founded upon the very nature of things. More than once you will be forced to recognize how difficult it is to distinguish, at the bedside, gout from rheumatism, above all in their chronic forms; and the name *gouty rheumatism*, which is often applied to those obscure cases which lie upon the confines of the two affections, seems to be an implied avowal of powerlessness in this respect.

However, gentlemen, we are deeply convinced that the two words *gout* and *rheumatism* correspond to two morbid types which are essentially distinct, and which should not be confounded with each other. This is what I hope to be able to prove to you in successively studying these two affections, so as to bring them, then, close together, and to make a comparison between them. It may be that we shall see them unite upon the ground of etiology—that is a point to which we shall refer later on; but, once established, they follow a parallel course, and never meet each other. They are, as an eminent pathologist has remarked, two branches springing from the same trunk. We accept, provisionally, this ingenious comparison, whose exactitude has not as yet been established; but let us at least be permitted to observe that, having once left the common trunk, these two branches bring forth very different fruit.

We shall commence this study, gentlemen, with the history of gout. Thanks to the recent works of which gout has been the subject, this affection is better known by us in very many respects than is rheumatism; besides, we consider it, from several standpoints, as a type of constitutional diseases—a model affection. By means of circumstances, unfortunately but too exceptional in medicine, we are here acquainted, past all question, with a *morbific material* whose presence gives rise to many and various phenomena; we hold a clue to guide us through the labyrinth, and we can follow, to a certain point, the logical series of symptoms which successively develop under its influence, during a long period of time. We are far from assuming that, to-day, we have traced gout to its ultimate essence; *first causes*, here as everywhere else, elude all our investigations and we may only flatter ourselves with having obtained one of the most important links in the chain: we have attained a knowledge of that vice of the humors upon which depend the principal phenomena that present themselves for our observation during the course of this disease. I hope, gentlemen, that the numerous details into which we are about to enter will not fatigue your kind attention. For, when in the presence of one of those diseases

which offer a material basis for our investigations—gout, syphilis, or saturnine poisoning,—we must probe the subject to the very bottom; thus, at least, one may hope to dispel, in part, the obscurity which still hangs over so many other questions in the domain of medicine.

First, however, a word concerning the general character of the diathesis we are about to study.

Gout is a chronic and constitutional disease, most often hereditary, and always connected with a particular dyscrasia—the presence of an excess of uric acid in the blood, constituting, in reality, one of the principal characteristics of the affection. It is undoubtedly true that the greater part of the morbid manifestations which give gout its particular aspect arise from that special condition; this, for example, takes place in the case of diseased articulations.

And you know, gentlemen, that, from an anatomical standpoint, gouty arthropathies are characterized by deposits of urate of soda, either in the interior of the joint or in the adjacent structures.

Independently, however, of these articular affections, and of that peculiar state of the blood, gout can give rise to numerous and varied visceral diseases, sometimes organic, and, again, purely functional.

There is even reason to believe that, in some instances—quite rare, however—the diathesis produces, during the whole period of evolution of the disease, only internal derangements of this kind, and never induces those external manifestations upon which we usually rely.

That, gentlemen, was what the ancients called *irregular* gout, in opposition to *regular* gout, which corresponds to the classical type of the malady.

But, even in the latter, we meet with visceral affections, sometimes manifesting themselves suddenly in the course of an attack (*retrocedent gout*—"*goutte rétrocédée, remontée*"), or in the intervals (*misplaced, alternating gout*—"*goutte mal placée*"); and sometimes, on the other hand, forming, by a slow, progressive, and, as it were, latent development, those deep organic lesions so often met with in individuals subject to gout (albuminous nephritis and fatty heart).

Thus, gentlemen, all that old nomenclature, bristling with fantastical terms, which the ancients applied to gout—all is unquestionably founded upon clinical observation; in the rigorous study of facts we shall discover apparent, regular gout; and irregular gout as masked, retrocedent, misplaced, etc. It would, no doubt, be useful to institute a reform in this language, which has now grown very obsolete; but we do not yet feel ourselves warranted in doing it, and so we must, perforce, continue to make use of the terms in vogue among the ancients, while we reserve our privilege to interpret their exact sense.

For this reason, gentlemen, you will hear us speak of *acute* and *chronic* gout. Now, gout is an essentially chronic affection, and can never be acute; still, these two names correspond to two of the principal phases of the disease.

Thus, a gouty patient will, at first, suffer from articular attacks, exhibiting all the appearances of an acute affection, which return periodically at more or less regular intervals. They may be limited to a small number of articulations, and more especially to the great toe; it is then acute *partial* gout. In acute *general* gout, which offers a great resemblance to rheumatism, all the articulations may be attacked, even the great ones; thus, for example, it is often seen at the same time in the knees, the elbows, and the wrists.

In the interval between the attacks, other affections which depend upon the gouty diathesis may make their appearance; such, for example, is the dyspepsia which so often torments the gouty; and such also is gravel, which, in certain individuals, manifests itself as alternating with the attacks of gout.

Quite frequently, during a paroxysm of acute gout, we notice the production of functional derangements, which may be attributed to retrocession of the disease; but visceral affections, which are connected with appreciable material lesions, are, on the contrary, very rare.

Chronic gout, which *may* supervene from the onset, only comes, in general, after several attacks of acute gout. The patient who used to enjoy long intervals of repose, sees the attacks becoming more and more numerous during the year, and coming nearer and nearer together. Their number increases without any diminution of the duration; and, in the end, they meet, they become superimposed—become, after a fashion, *subintrant*, to make use of a term borrowed from the history of paludal fevers;[1] indeed, the patient is the prey of almost continuous pain, with alternate exacerbations and remissions.

Corresponding to these permanent symptoms are permanent lesions, which are, at first, in the joints, and afterward occur in the internal organs; and it is in chronic gout pre-eminently that we find those grave visceral affections which, in general, are the direct causes of death. Besides, when the disease is prolonged, we finally see that cachectic condition supervening which ordinarily terminates all great constitutional affections. Then do dropsy, anæmia, and marasmus develop; and then it is that the patient sinks into an almost complete state of atony—when nature seems no longer to answer to the therapeutic measures which are exhibited to oppose the progress of the disease.

Having stated these preliminary ideas on the subject, we shall enter directly upon the history of gout, commencing with the study of those anatomical alterations which are its necessary attendants; and since, in this general disease, the blood-condition seems to be the all-controlling element, we shall first invite your attention to that most important topic.

Pathological Blood-conditions of Gout.

Since the time when Scheele discovered lithic acid, which we to-day call uric acid, many authorities have thought that this principle might develop, during the course of gout, in the fluids of the economy. Wollaston was the first to prove that gouty concretions were composed of alkaline urates; and since then Forbes-Murray and Holland in England, Jahn in Germany, and Rayer and Cruveilhier in France, have expressed the opinion that in gout the blood must contain uric acid. But the honor of furnishing the positive demonstration of this fact belongs to Garrod.[2]

Normally there exist traces of uric acid in the blood; but during an attack of gout the blood may contain 0.05 gramme to 0.17 gramme of it in 1,000 grammes—($\frac{1}{2000}$ to $\frac{1}{6000}$). But, to make manifest this proportion, it is necessary to have recourse to very delicate chemical manipulations, which are beyond the domain of the clinic.

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[1] "Febris subintrans"—a fever where paroxysms succeed one another without intermissions.—L. H. H.

[2] Medico-Chirurgical Transactions, 1848.

There is a simple process, one whose application is much easier, which, though not indicating precisely the quantity of uric acid in the blood, enables us to establish its presence therein. Put about five grammes of serum in a clock-glass (not in a watch-glass, inasmuch as its curvature is too marked), add a few drops of acetic acid, and then put in a piece of thread. Allow the liquid to remain in a dry place from thirty-six to forty-eight hours; then by the aid of the microscope you can determine the presence of rhomboidal crystals, which encrust the thread immersed in the liquid. These crystals are composed of uric acid.

In order to obtain this result, certain precautions must be observed. First, care must be taken that the serum is fresh, for the presence of albuminoid matter develops a sort of fermentation in it; the uric acid then decomposing into oxalic acid, urea, and allantoin, just as in the presence of puce oxide of lead.[1]

Too much drying of the serum must also be avoided, since in that case crystals of ammonia and magnesia phosphate would form and appear as very beautiful vegetations. But, as this is quite a soluble salt, it is sufficient to add a little water to the preparation in order to dissolve it; and then masses of rhomboidal crystals, composed entirely of uric acid, will be seen making their appearance.

This process, though not sensitive enough to indicate the trace of uric acid which exists normally in the blood, is yet amply sufficient for all practical needs; in reality it reveals the presence of *one sixty-five-thousandth* part of uric acid in the blood (Garrod).

When blood is not at your disposal, it may be replaced by the serosity of a blister, which will give the same reactions, provided care has been taken not to apply this revulsive at any place invaded by the gouty inflammation; for all phlogistic action causes uric acid to disappear.

It is very easy to understand the clinical importance of this process; in many cases it is an excellent means of diagnosis. It also enables us to discover under what circumstances an excess of uric acid is produced in the blood. This phenomenon exists permanently in cases of chronic gout; but its intensity is augmented during a paroxysm, to fall thereafter below the limits previously marking it. In acute gout it does not occur in the interval between the attacks, at least at the commencement of the disease (Garrod),[2] but manifests itself anew some time before the onset of the attack. Lastly, in cases of ab-articular gout, we notice the appearance of various symptoms which seem to be connected with the same condition of affairs, since analysis reveals the presence of uric acid in the blood.

Now, on the contrary, acute articular rheumatism (Garrod), or chronic rheumatism (Charcot), is never connected with that particular dyscrasia; and here we find a useful element of diagnosis in doubtful cases, since it then is quite sufficient to apply a blister to the patient or draw a few grammes of blood from him, in order to feel assured whether it is to gout or rather to rheumatism that the observed phenomena should be ascribed.

However, this excess of uric acid must not be considered as a pathognomonic symptom of the gouty diathesis; such a phenomenon occurs in Bright's disease and in cases of lead-poisoning. It is, nevertheless, probable that this particular condition induces a predisposition to gout; at least it seems to explain the frequency of this affection in the lead-workers of London (Garrod).

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[1] *Puce oxide:* per- or binoxide.

[2] See Reynolds: A System of Medicine. Article, Gout. London, 1866.

The presence of uric acid in the *humors* of the gouty is likewise revealed by the composition of various fluids, either normal or pathological. I have found it in the cerebro-rachidian fluid, and Garrod met with it in the serous effusions into the pleural and pericardial sacs. It is not known with certainty whether it exists in the intestinal secretions, but it is found in the pustular liquid of eczema (Golding Bird), and in the white, powdery substance which sometimes forms upon the skin of gouty patients; this powder is composed essentially of urate of soda (Petit, O. Henry).[1] In every case it is very certain that sweat, whether spontaneous or induced, does not contain a trace of it (Garrod, De Martini, Ubaldini).[2]

We have yet to ask ourselves the question whether, in gout, the blood does not also present other alterations in its chemical constitution. Although this part of the subject is still quite obscure, it nevertheless seems to have been established:

First.—That the proportion of blood-globules is maintained at the normal standard in gout, thus evidently contrasting with rheumatic anæmia; while in chronic gout there is, in the long run, a diminution of the globules—this is gouty anæmia.

Second.—That in acute gout there is augmentation in the fibrin; the bleedings at least, are buffy.

Third.—That in chronic gout the albumen of the blood diminishes if there be a diseased condition of the kidneys, in which case an excess of urea will be discovered.

Fourth.—That the alkalinity of the blood is always diminished, and this it is which seems to favor the production and deposition of concretions.

Fifth.—Lastly, that sometimes the blood contains traces of oxalic acid.

As a complemental part of this study we should examine into the state of the urine in gout; for the question is, whether uric acid is found therein in a much greater proportion than usual, as has been claimed; or whether, on the other hand, it is in much smaller quantity, as careful modern investigations seem to have demonstrated.

In order to obtain a reliable solution of this question, it is not only necessary to estimate the proportional quantity of uric acid contained in one specimen of urine, but to find out the total amount of uric acid eliminated by the kidneys in the interval of twenty-four hours, and this, not only during a single day, but for several days, inasmuch as the excretion of uric acid by the kidneys is intermittent.

It is absolutely necessary, then, to have recourse here to a methodical analysis; and one ought to remember that the presence of a free acid in the urine, or in a specimen where there is not an abundance of the watery part of this fluid, will be accompanied by the formation of those sediments to which generally such an exaggerated degree of importance is attached.

It is after having directed attention to all these sources of error that Garrod arrives at the following results:

In acute gout, during the fit or paroxysm, the urine is scanty and dark in color, but the quantity of uric acid excreted in twenty-four hours is almost always considerably less than in the normal state of affairs (0.25 gr. instead of 0.50 gr.). There is, thus, a diminution in the excretion of this product coincident with an augmentation of its proportion in the blood.

In the interval between the attacks, the urine was not examined; still, we shall find that gravel is frequent, as likewise are the crystallized de-

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[1] Journal de Pharmacie. October, 1841.
[2] Union Médicale, No. 40, p. 24. April, 1860.

posits of uric acid formed before micturition (Rayer); but the occurrence of this phenomenon is not sufficient to prove that there is an actual excess of uric acid, either in the blood or in the urinary excretions.

In chronic gout the tendency to diminution increases more and more. During an attack the urine is pale, and copious in amount; so long as the disease remains apyretic, it does not form sediments upon being cooled, and merely *traces* of uric acid are found in it. Still, from time to time there are *unloadings* (*décharges*), during which the urine contains a considerably greater quantity of this product.

In the intervals between the attacks these characteristics persist: albuminuria is frequently observed, and sometimes the urine contains fibrinous cylinders.

To sum up, gentlemen, it is clear that, under the influence of the gouty diathesis, there is a superabundance of the urate of soda in the blood and in the *humors*, to speak in the medical language of ancient days; urate of soda likewise constitutes those articular deposits which at all times have been markedly noticed in the gouty. But this excess of uric acid does not manifest itself by an augmentation of the renal secretion, but seems, on the contrary, to coincide with a faulty elimination.

LECTURE IV.

PATHOLOGICAL ANATOMY OF GOUT.

Summary—Local Changes in Gout—Condition of the Articulations—Diarthrodial Cartilage—Deposits of Urate of Soda occupy by Preference those Tissues Deprived of Vessels—Condition of the Synovial Membranes and the Ligaments—Tophus (Chalk-Stone); its Composition—Inflammatory Phenomena—Dry Arthritis—Ankylosis—Place of Election of Gout: Articulations which it may Invade—Peri-Articular Tophaceous Concretion—Deep-seated Cutaneous Concretions—Tophus of the External Ear—Enumeration of the Principal Points where a Tophus may Form.

Gentlemen:—In the last lecture we have seen that, at all periods of its evolution, gout coincides with an excess of uric acid in the blood.

To-day we are going to show you that the local changes in this disease arise, for the most part, from the direct consequences of this general alteration, and that the deposits which are met with deep within organs or tissues are almost always formed of urate of soda.

The knowledge of the changes to which we invite your attention is not confined to modern days. Long ago it was known that, in gouty patients, *tophi* and chalky *deposits* formed around the joints; but these were looked upon as exceptional occurrences, and were thought to be peculiar to the gravest and most inveterate cases. It was reserved for Garrod to show that *the slightest attack of gout* leaves an indelible imprint upon the tissues it invades, and that the latter are forever stamped with its seal.

Let us begin by studying what occurs in the diseased articulations: there we shall see gout manifesting itself by truly peculiar and constant anatomical characteristics.

I.—From the very first attack deposits of urate of soda form in the articular[1] (*diarthrodial*) cartilage;[2] they occupy the most superficial portions of the cartilage, and are lodged either in the space between the cells, or in their very interior—a fact which Cornil and myself have established. They are generally located about the centre of this free surface, as far as possible from the insertions of the synovial membrane, which stops, as you know, at the circumference of the articular cartilages.

It is not difficult to comprehend the reason of this singular choice. The points accessible to the circulation are the least liable to the formation of these deposits which occupy preferably those tissues which are deprived of vessels; now, synovial membranes and bone possess an eminently vascular structure, and thus gouty concretions form on the surface of the cartilage so as to be separated from the bone, and form at the centre of this surface so as to be removed from the synovial membrane.

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[1] Garrod: On Gout, p. 211. London, 1863.

[2] Articular, also called diarthrodial cartilages, and "cartilages of incrustation" (cartilage d'encroûtement).—L. H. H.

At a more advanced period of the disease, when the chronic stage has succeeded the acute, the synovial membrane itself then suffers invasion, and the appendages of the fringe-like processes at the margin of this membrane, being less rich in vascular supply, are the first to be attacked ; later on the synovial membrane itself presents incrustations. It is then that deposits form in the epithelial cells, according to Professor Rouget ; and the whitish sediment or mud—(*boue blanchâtre*)—which is sometimes observed in gouty articulations is nothing but urate of soda proceeding from epithelial desquamation.

We know, indeed, that the ligaments themselves at times participate in this process of incrustation. But it is not even here that the pathological process ceases ; it may go farther and invade parts extraneous to the articulation; the tendons and the synovial sacs may become the seat of it, and when a concretion develops in the adjacent cellular tissue it receives the name of *tophus*, or chalk-stone. Sometimes, you know, it attains a considerable size. These extra-articular lesions, however, which correspond to a more advanced state of saturation, are always secondary to those changes in the diarthrodial cartilages, which may exist alone, but which can never be absent when deposits of urates have occurred in the circumference of the articulation. At least, we know of no case which forms an exception to this rule.

And now let us see what is the composition of the material that constitutes these deposits. Examined with the naked eye, it appears amorphous, and resembles plaster-of-Paris ; but, viewed microscopically, it seems to be entirely formed of needle-like crystals. It is true that you sometimes find masses of amorphous matter disseminated throughout the affected cartilage ; but Garrod claims that by means of the polariscope you can establish the fact that these agglomerations are themselves possessed of a crystalline structure.

When acetic acid is employed, rhomboidal crystals of uric acid are produced, and it is by means of this reagent that we can prove the presence of depositions in the interior of cartilage-cells. But we possess other means for determining the chemical composition of these incrustations : if the affected cartilage be treated first with cold water, then with alcohol, and next with hot water, it becomes perfectly transparent, and the reagents that have been used in this washing deposit, upon evaporation, crystals of pure urate of soda. These crystals, when incinerated, produce carbonate of soda; treated with fuming nitric acid, and then with ammonia, they give rise to purpurate of ammonia, or *murexide*, whose color is so characteristic.

But we shall not further urge the chemical part of the question ; it will be sufficient to remark that the cartilage, when thus rid of its incrustations, presents a perfectly normal structure, with no change visible either under the microscope or to the unaided eye. At least, this is the general rule.

As for the liquid—often muddy—contained in some instances within the articular cavity, it quite often has an acid reaction, presenting microscopically epithelial *débris* and needle-shaped crystals.

II.—Let us now point out some other lesions, which, though they are not constant, none the less merit a detailed description.

When a gouty articulation is opened just after an attack, the synovial membrane is almost always found red, injected, and vascularized; but while these phenomena never go on to suppuration, an excess of fluid is, however, often found within the articular cavity.

In cases of inveterate gout all the lesions of a dry arthritis may be met with at the diseased point; and consumption of the cartilages ("*usur*"), secondary ulcerations and osseous swellings, have all been remarked by various observers. Indeed, I have myself seen some cases where they were present; but they are exceptional occurrences, whose nature is as yet not well understood, but which deserves a very attentive study.

Do you see in these peculiar cases a kind of transition between gout and rheumatism? Can it be that they are the results of a kind of combination of these two diatheses? Or is it only a question of a simple complication?

This is a question whose answer, it seems to us, ought to be postponed.

Finally, ankylosis may be the result of the changes which have just been described: sometimes it amounts to only a simple rigidity, the result of incrustation of the ligaments; but true osseous ankylosis is also met with, as has been remarked by Garrod and by Ranvier. Indeed, this may come from the very first attack, as Todd and Professor Trousseau have pointed out.[1]

III.—Gout does not impartially choose its seat from all the joints, as was well known in the days of remotest antiquity, for the metatarso-phalangeal articulation of the great toe enjoys the unpleasant prerogative of most frequently drawing down upon itself the manifestations of this disease; then come the fingers, followed, after a long interval, by the knees and the elbows. The hip- and shoulder-joints are usually spared.

Sometimes, however, the great toe is not attacked by the gout while other articulations are being invaded; and this is a fact of great practical importance, since it allows us to understand why general acute gout sometimes presents such a strong resemblance to acute articular rheumatism, and to explain how it is that certain observers have been led to confound[2] the two.

It is evident, for example, that an attack of acute gout, occurring simultaneously in the knees and wrists, would be very difficult to distinguish, at the bedside, from a purely rheumatic affection.

Among the rare and exceptional cases we may mention those where gout attacks the vertebral column, the temporo-maxillary articulation (Ure), the arytenoid cartilages (Garrod), and finally the ossicles of the ear (Harvey): this results in a new species of deafness.

IV.—From this necroscopic study we shall deduce a number of considerations, the importance of which, from a clinical standpoint, is incontestable.

First.—At once let us observe that the incrustation of cartilages is inseparable from articular gout, and seems to begin with the first attack.

Second.—In a gouty patient the diseased joints alone present this lesion of their cartilages, and now and then it is found only in a single articulation.

Third.—This incrustation of urate of soda goes on independently of the paroxysms or attacks, and in the interval between them it may not reveal itself upon the exterior of the joint by any appreciable deformity.

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[1] Todd: Practical Remarks on Gout, p. 45. London, 1843. Trousseau: Clinique Méd. de l'Hôtel-Dieu, vol. iii., p. 328.

[2] In the original it reads: "amenés à les comprendre." I have taken the liberty to substitute *confondre* in correction of what I deem a printer's error, the context certainly pointing to *confound* instead of *comprehend*. —L. H. H.

Fourth.—This lesion is peculiar to gout, and never occurs in articular rheumatism, whether acute or chronic.

There yet remain the questions: What relationship subsists between an attack of gout and the formation of a deposit? Is the latter phenomenon the cause or the effect of the symptoms which accompany it? This last is a difficult question to answer, and we shall reserve its discussion for another time.

V.—We have seen that deposits of urate of soda form upon the exterior of diseased articulations. They are met with: 1st, in the tendons, and more particularly in the tendo Achillis; 2d, upon the periosteum, but never in osseous tissue; 3d, in the serous sacs (the olecranon and patella's); 4th, in the subcutaneous cellular tissue; and 5th, even in the deep layers of the skin. The last two points especially deserve consideration.

The subcutaneous depositions which form in the vicinity of joints constitute a very important part of the symptomatology of chronic gout, for they often manifest themselves during life. They are known by the name of *tophi*, *tophaceous concretions*, or *chalk-stones*—terms which are very frequently misapplied. They are exclusively given to the peri-articular collections of urate of soda, and ought never to be employed to designate the osseous tumors of chronic articular rheumatism.

In the earlier stages of their development these chalky masses have a soft and doughy consistence; later on they harden, and acquire a certain degree of solidity. From a chemical point of view, they are composed of urate of soda, mingled with the urate and phosphate of lime. Microscopically, they present very fine crystallized needles.

Their favorite seat is in the hands and upon the surface of extension; but they are equally found about the great toe and at other localities. They are ovoid tumors with an irregular surface, sometimes being sessile, sometimes having a pedicle, and at times attaining a volume equal to that of a pigeon's egg; they are in the immediate vicinity of the joints, without exactly resting upon them; being movable laterally, they do not precisely reproduce the form and the contour of the osseous heads in juxtaposition with them. They exert a lateral pressure upon the joints which does not always deform them; they present no symmetry in their mode of distribution; the skin covering them is shiny, sometimes being of a dull white color, and with transmitted light you may be able to see the subjacent deposits.

These various properties enable us to distinguish the *tophus* from those characteristic deformities of nodular rheumatism which we shall study later on in the course. But we must not overlook the fact that sometimes very difficult cases occur, in which there is an angular deviation of the fingers analogous to that met with in chronic rheumatism. Here the consideration of the articular deformities alone will not suffice to establish a diagnosis, if no external *tophi* exist. You must, in that emergency, rely upon those general or local phenomena which, taken as a whole, characterize the gouty diathesis; and you may even meet with cases where it will be necessary to have recourse to a chemical examination of the blood before arriving at absolute certainty in your diagnosis.

VI.—These concretions which form in the deep layers of the skin, also offer, from a practical point of view, a special interest.

The deposits in the external ear described by Ideler, Scudamore, and

Professor Cruveilhier stand in the foremost rank ; and Garrod has shown how great an advantage may be taken of them, in respect to the clinic.

The seat of these little concretions is generally upon the edge of the helix, but they may occur in the anti-helix or on the internal surface of the auricle or pavilion of the ear. They pass through three stages of development: at first soft and pulpy, they next become hard, and form small, whitish masses ; finally they may fall off, leaving behind them a small cicatrix whose existence may be proven when the tophus itself has disappeared.

Of 37 cases Garrod found external tophi in 17 ; upon the ear alone in 7 instances, and upon the ear, and near the joints, 8 times ; once only on the line of an articulation was a tophus found, without a coincident, similar deposit in the ear.

These decisive indications sometimes become manifest very early ; I have been able to predict in advance, simply by the presence of a tophus in the ear, the outburst of gout in a dyspeptic patient, in whom, nevertheless, articular symptoms had never appeared up to the time when he consulted me. Garrod witnessed the formation of these concretions in one of his patients five years before the appearance of any symptoms in the joints.[1] In this way one readily understands what importance they have from a diagnostic standpoint.

When concretions upon the external ear are wanting, the following parts should be examined: 1st, the eyelids ; 2d, the alæ of the nose ; 3d, the cheeks ; 4th, the palms of the hands ; 5th, the corpora cavernosa.

Upon all these points cutaneous depositions have occurred identical with those which we have just described.

It now remains for us to speak of the pathological anatomy of visceral gout, and to that we shall devote our next lecture.

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[1] These exceptional cases do not, however, invalidate the general rule. Articular symptoms almost always precede the formation of these external deposits.

LECTURE V.

PATHOLOGICAL ANATOMY OF VISCERAL GOUT.

Summary.—Retrocedent Gout; *Functional Lesions* of Gout—In most Instances after an Autopsy has been made, these seem to arise from Material Changes—Organic Lesions most frequently met with in the Viscera of Gouty Subjects—Fatty Degeneration of the Heart—Atheroma of the Aorta—Bronchial Lesions—Gouty Nephritis: Its Two Distinct Varieties—Gouty Kidney of the English: Lesions Corresponding to this Designation—Deposits of Urate of Soda—Bright's Disease—Interstitial Nephritis.

Changes Analogous to Gouty Changes in Animals—Do not Exist in Mammals—Occur in Certain Birds—Similar Lesions in Reptiles—Experiments of Zalesky—Results of Ligation of the Ureters in Different Animals.

GENTLEMEN:—If gout were a more common affection in our hospitals, we would probably be better acquainted with the pathological anatomy of the visceral lesions that may arise from this disease. But, as you are aware, the opportunity of making an autopsy of a gouty subject very rarely presents itself in France. The English authors fare much better in this matter, and they have given us some very interesting details concerning the subject. It may be stated, however, in a general way, that little is known of the whole question, at least with respect to the anatomical lesions. Gouty nephritis alone stands as an exception to this rule; and for this reason it behooves us to study it with particular care, after having rapidly outlined the state of our knowledge touching the other visceral manifestations of gout.

I.—We shall first consider those sudden modifications, occurring in the course of gout, which have received the name of metastases or retrocessions.

In such a case one would evidently suppose, upon a first view, that it is only a question of mere superficial lesions, especially when the symptoms are not followed by a fatal issue; it does not seem probable that the diseased organ could be the locus of deep-seated changes, and one would rather be disposed to class derangements of this kind under the head of *functional lesions*. But here the stamp of pathological anatomy is almost completely wanting, by very reason of the rapidity with which these symptoms disappear in the majority of cases.

Still, we do not always find things happening in this way; death is sometimes the result, and one may be called upon to make the autopsy. In such cases organic lesions have often been discovered, and, in this regard, we possess several observations which are worthy of credit. Certain of these cases, which were accompanied by gastric symptoms of gout, came under the observation of Dietrich, Perry, and Budd. The patients succumbed with the classical symptoms of gout in the stomach; and an œdematous swelling of the submucous cellular tissue of the organ was discovered, along with more or less marked changes within the mucous membrane itself.

In other cases, where the patients rapidly sank during the course of a gouty attack or fit, the ordinary lesions of a cerebral hemorrhage have been discovered, and sometimes even rupture of the heart has occurred.

If it were allowable to judge in view of these examples, we would be induced to believe that, even in those cases where functional derangements manifest themselves upon the occasion of gout, they nevertheless correspond to organic changes less superficial than generally supposed.

But there are other lesions which seem to be connected with this disease, which have been met with in subjects who have suffered from either acute or chronic gout, when an intercurrent disease, or the progress of the gout itself, has induced a fatal termination. These we shall hastily review.

A.—The muscular walls of the heart are often affected with fatty degeneration. S. Edwards, Lobstein, and some other observers, have found urate of soda in valvular concretions, although Garrod contests the accuracy of this statement.

B.—The aorta is often the seat of atheromatous change; moreover, urate of soda has been discovered in its walls by Bramson, Bence-Jones and Landerer.

C.—Bence-Jones has asserted the experience of urate of soda in the walls of the bronchial ramifications.

D.—Up to the present time no special change has been ascertained to to be present in the brain, meninges, or in the encephalic arteries.

E.—The kidney-changes in gout, ordinarily described under the head of gouty nephritis, are to be divided into two varieties.

In the *first* place we find the disease described by Rayer under the name of gouty nephritis, which may be properly denominated *gravel of the kidney*. It presents the characteristics of chronic interstitial nephritis, but is pre-eminently marked by infarctions of fine gravel and uric acid, the latter sometimes in a crystalline condition, although larger-sized gravel may also be present. These deposits occur; 1st, on the surface of the kidney and deep in the cortical substance; 2d, in the mammillary elevations and papillæ; 3d, in the calices and infundibula; upon the last-named part the concretions are generally large in size.

These changes may also occur apart from articular gout, but they are undoubtedly of very common occurrence in this affection.

In the *second* place we have what is properly called *gouty* nephritis; this is the *gouty kidney* of English writers. Signalized by De Castelnau in 1843, it has been excellently described by Todd and Garrod. Anatomically it is characterized:

First.—By urate of soda infarctions in the form of white streaks (*traînées blanchâtres*), which are found in the tubular substance (never in the cortical portion), and, in some cases, in the pyramids. Microscopically, they appear in the form of crystalline needles, whose seat, according to Garrod, is in the spaces between the uriniferous tubules; although we believe we have proved that their starting-point is in the very canal of the uriniferous tubules which are obstructed by them.[1]

Second.—It is characterized by concomitant changes within the kidney, which correspond to the ordinary lesions of Bright's disease.

At first there exists a parenchymatous nephritis,[2] which may appear in

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[1] Charcot and Cornil: Mémoires de la Société de Biologie. 1864.

[2] Loc. cit.

one of two different stages. In the *first* stage the kidney preserves its ordinary size, but the cortical substance becomes thicker and presents a yellowish tint. The Malpighian corpuscles are injected, the uriniferous tubules are filled with epithelial cells which are distended, opaque, and full of fatty or proteid (albuminoid) granulations.

In the *second* stage you find atrophy of the cortical substance and that granular condition of the kidney belonging peculiarly to Bright's disease.

But, besides this parenchymatous nephritis, you also meet with the interstitial nephritis corresponding to the *gouty kidney* of English writers.

This is especially characterized by a thickening of the intertubular connective tissue, and a proliferation of nuclei (cells, *noyaux*). The volume of the kidney is diminished, and its surface is wrinkled, granular, and red. The cortical substance is markedly atrophied. In this disease the kidneys have never been seen in a waxy condition.

According to Garrod, these changes have existed in all cases of inveterate gout where an autopsy has been made ; and they may be present very early in the disease, having been observed after the seventh or eighth attack. In one case recorded by Traube, the symptoms of the renal affection occurred only one year after the first manifestations of gout. This was undoubtedly a visceral form of the disease.

Although the changes in the parenchyma of the kidney differ in no way, excepting in the deposition of urate of soda, from those which exist in ordinary Bright's disease, yet the symptoms belonging to this gouty albuminuria are remarkable on account of their mildness and the slight degree of intensity which they present. But I do not wish to dwell upon this point, which belongs to symptomatology.

To this kind of causes we may assign many symptoms which very frequently figure in the train of those accompanying gout. In this way *dyspepsia* is often aggravated, if not wholly caused by that pathological condition of the kidney ; and again, œdema is frequently a consequence of it. Uræmia is seen in the convulsive or comatose form in gouty patients, and evidently arises from this state of the kidneys. Cerebral apoplexy, cardiac hypertrophy—both of these may be reckoned among the remote consequences of the renal lesion.

II.—This, gentlemen, finishes the sketch of the anatomical lesions which characterize the gouty diathesis. But, before leaving the subject, I believe it profitable to look at certain facts concerning *comparative pathology* and *experimental pathology*, which seem to me of a nature to illumine with a powerful light those questions which we have already raised, and those also which we shall next consider.

Can gout occur in animals ? And, supposing that it can exist in them, does it produce results comparable with those it causes in the human species ?

In a recent work upon comparative pathology, Gleisberg answers in the negative. He rightly observes that most of the affections which have been designated by this name ought to be classed under the head of chronic rheumatism. At all events, it is incontestable that a disease strikingly analogous to gout in man, develops, and is characterized, like the latter, by deposits of urate of soda in the various tissues of the body.

We do not find our points of comparison among animals, as you would be led to suppose from the analogy, but in birds and certain reptiles kept in captivity and placed under special conditions.

You will be less astonished at finding, in these two great classes of animals, disease presenting some point of resemblance to gout, when you re-

member that the work of disassimilation in them does not produce urea, but rather urate of ammonia, as Davy and other chemists have shown.

Gentlemen, authors of the various treatises upon ornithology will tell you that, in certain birds, there may exist lesions closely analogous to those of gout. Aldrovandi teaches us that, in the *falconidæ*, tumors made up of masses of gypseous matter are liable to form around the toes. This is an incurable condition.

Analogous phenomena occur in parrots. Bertin of Utrecht found, in the *Psittacus grandis*, urate-tumors in the vicinity of the joints, and similar infarctions in the articulations and in the kidneys.

Lesions of a similar character have been pointed out in the case of reptiles. Pagenstecher observed the kidneys and joints of the *Alligator sclerops* affected in this way. In *ophidians* these changes may exist in the kidney, and in the tortoise Bertin has likewise found both articular and renal lesions.

It is curious thus to meet, in animals but remotely related to man, these lesions, which, in all respects, are so strikingly analogous to those of gout. But a still more remarkable fact is that these lesions may be artificially induced by means of physiological experimentation. This is the result of an interesting work published at Tübingen, by Zalesky.

This experimentalist ligated the ureters of chickens, geese and adders (*Coluber natrix*). The first morbid phenomena appeared in from twelve to fifteen hours after the operation. Life was prolonged over two or three days, and, after death, urate of soda was found in the following viscera:

First.—The kidneys exhibited it within their tubules, but not in the cortical substance. The ureters likewise contained it.

Second.—The lymphatics, serous membranes, the cellular tissue, and the capsules of all the organs, were impregnated with it.

Third.—The stomach-follicles contained a remarkable quantity of it.

Fourth.—It was found in the valves of the heart.

Fifth.—The joints exhibited, both *intra* and *extra*, a considerable accumulation of this product.

There was no deposit of urate of soda in the muscles, but the muscle-juice contained a great deal of uric acid.

The brain and its membranes seemed to be wholly exempt.

Concerning the fluids of the economy, the blood contained marked quantities of uric acid, and after death clots were found composed of alkaline urates.

Finally, the gall-bladder contained an enormous amount of urate of soda, a new coincidence with human pathology; for in man, according to Frerichs, calculi composed of urate of soda are sometimes found in the gall-bladder.

In order to accept unreservedly these data, the experiments must be renewed and varied; the effects produced by the ligation of a single ureter, for example, must be studied, in order to see whether, after a certain lapse of time, the same results will not be produced. It is none the less true, however, that Zalesky's experiments possess great interest, and deserve to be seriously considered.

Gentlemen, though this excursion into the domain of comparative pathology may seem a little unusual to you in a clinic, you must not be astonished at the importance which we attach to these facts. We shall meet them again at the time when we shall endeavor to argue a physiological theory of gout.

LECTURE VI.

SEMEIOLOGY OF GOUT.—URIC ACID DIATHESIS.—ACUTE GOUT.—CHRONIC GOUT.

Summary.—The Two Principal Forms of Gout; Acute and Chronic Gout—Gout always Essentially a Chronic Affection; but the Acute Attack is very Different in Appearance from the Permanent Condition—Uric Acid Diathesis—Symptoms which Collectively Characterize it—Urinary Secretion less copious, but richer in Solid Matter—Microscopic Gravel—Acute Gout—Prodromata—Invasion of Articular Pains—General Symptoms—Principal Characteristics of Acute Gout—Secondary Phenomena—Deviations from the Regular Type—General Acute Gout—Asthenic or Atonic Gout—Return of the Attack or Fit—Imperceptible Transformation of Acute into Chronic Gout—Gouty Cachexia—Gravity of Intercurrent Diseases—Chronic Gout following Acute Gout—Gout Chronic from the Onset—Development of a Tophus.

GENTLEMEN:—Heretofore the anatomical changes of gout have exclusively occupied our attention. We have studied the alterations which both the fluids and solids of the economy have undergone under the influence of this disease, and it now remains for us to submit to your view the symptoms arising from the existence of such changes.

At the beginning of these lectures we established the fact that, whether acute or chronic, gout appears in one of two principal forms, each of which merits a special description. It must not be forgotten, however, that in every case gout is an essentially chronic affection. The fact is, the first attack is but the primitive manifestation of a constitutional condition, which after this initial paroxysm may remain in a latent condition during a certain number of years, but which sooner or later will reveal itself by new symptoms. If there do exist cases of transient gout marked but by a single attack, they are phenomena of a very exceptional kind, and cannot invalidate the general rule.

Still, the aspect of an acute attack is so very different from that presented by the disease when in its permanent condition, that we are forced to respect these expressions perpetuated by usage, and which, in spite of their inexactitude, undoubtedly correspond to real occurrences.

We are about to study in succession, therefore, acute and chronic gout, in this way conforming to the universally accepted tradition. But before entering upon the subject, it is proper to invite your attention to a morbid condition which sometimes manifests itself before the appearance of articular symptoms, and which, in those who are already subjects of gout, quite often fills in the intervals between its attacks. This you have already recognized as the uric acid diathesis. Let us consider, then, this general condition for a few moments, a condition which is known chiefly by means of English writers.[1]

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[1] Todd: Practical Remarks on Gout, etc., London, 1843. Budd (George): On the Organic Diseases of the Stomach, etc. London, 1855.

Uric Acid Diathesis.

The chief occurrence—the one that rules the whole situation here—is a peculiar dyspepsia, whose ordinary symptoms are flatulence and distention of the stomach, and consequently acidity of the stomach and pyrosis. At the same time there is a bitter taste in the mouth, with dryness of the tongue and supervention of the saburall state. Constipation is almost always present in these cases.

The liver also seems to participate in the derangements of the digestive apparatus, and, as it is frequently swollen, it passes below the free margin of the ribs. A slightly jaundiced hue is sometimes observed, and the stools, besides being rather scanty, become gray and discolored.

Quite pronounced nervous phenomena accompany this dyspeptic condition, and may even exist apart from it. You notice spontaneous attacks of lassitude, with despondency and headache. Sleep is disturbed, and the patients frequently pass into the hypochondriacal state. In women this condition may be complicated by manifestations of a distinctly hysterical nature.

In others, cardiac palpitation and a bronchial catarrh may occur in addition. And these phenomena which have not, *per se*, any great importance acquire a greater significance on account of the other circumstances accompanying them. But what pre-eminently characterize this particular condition of body are the exacerbations it periodically undergoes, and its subjection to a most evident influence from errors in diet—a trait in common with gout, whose importance should not escape us.

What is the state of the urinary secretion in such a case? In a general way the urine is scantier, but richer in solid matter; it is diminished in quantity, but very acid and high-colored; it is loaded with sediment, which usually forms after voiding of the urine, but which may be already formed within the bladder. In such cases crystals of uric acid are very frequently met with in the urine; this constitutes Rayer's *microscopic gravel*.

When the uric acid diathesis appears before the articular phenomena of gout, one may demonstrate, in some instances, the existence of an excess of uric acid in the blood from that moment. And, besides, the whole series of symptoms we have just been describing occur especially during the interval between the attacks, when rheumatoid pains will manifest themselves in groups of muscles, and in those articulations having a predilection for inflammation. These pains appear abruptly in the form of twinges, and during the absence of articular inflammation, they constitute one of the most characteristic evidences of gout, sometimes, indeed, preceding the outburst of the disease.

Gentlemen, there are, undoubtedly, cases in which this condition of affairs may remain unaltered, and in which the predisposition giving rise to the uric acid diathesis never receives the last factor in this morbid process, just as gout develops, in certain subjects, at the initial attack or paroxysm; but the general rule is, that upon this pathological foundation is developed gout properly so-called, and this development of gout we shall now commence to study.

Acute Gout.

Let us suppose that a first attack of gout is on the point of breaking forth in one who, up to this time, has never felt a touch of this disease. In the majority of cases special prodromata announce the coming of the attack;

sometimes there will be an extreme exacerbation of all the symptoms resulting from the uric acid diathesis, which I have just been describing to you; and, at other times, on the contrary, there will be an abnormal feeling of well-being, a peculiar excitation. Finally, in a few cases you may observe the production of phenomena wholly unknown to the patient's experience, as, for example, an angina, a sciatica, or muscular pains. Still, you must recollect that in a certain number of cases there may be a complete absence of prodromata, the attack then commencing abruptly in an absolutely unforeseen manner.

The occurrence of articular pains is rapid and violent, almost always taking place during the night. The patient suddenly feels a characteristic pain, likened by some to a bite, and by others to a blow from a cudgel; many imagine that they have suffered a sprain, and on the first view the diagnosis is somewhat difficult. The most frequent seat of these sensations is the metatarso-phalangeal articulation of the great toe. Soon redness and swelling appear at the point attacked; there is enlargement of the veins of the affected member, which assumes a purple color, and, according to Gairdner, is now and then covered with ecchymoses. Fluctuation, sometimes apparent, sometimes real, is developed at the same time; and when real is caused by the presence of an excess of fluid within the articular synovial membrane.

The general symptoms accompanying these local manifestations are fever, irregular chills, nervousness, and great irritability; and, finally, a marked diminution in the quantity of the urine, which upon cooling deposits a very copious sediment.

Toward morning the pain and most of the other symptoms remit, to reappear that evening or during the night. This condition continues for five or six days should medicine have been administered, and for eight or fifteen should no medicines have been employed (Garrod). There is, then, a kind of chain of short attacks linked one to another, and separated by intervals of remission.

In the first days of the attack marked œdema appears in the inflamed parts, soon extending over the whole member, and pitting on pressure. The decline is characterized by superficial desquamation. Ultimately everything is restored to a normal condition and the patient enjoys a respite, only to be broken by the next crisis.

Thus you see that, to sum up, the salient features of acute gout are the following:

First.—The abrupt invasion and the special character of the pain. A Frenchman, quoted by Watson,[1] comparing the sensation which he had so often experienced with the effects of great pressure, said that rheumatism was the first turn of the vise; but, give it another turn, and you had gout.[2]

Second.—Œdema of the member at the commencement of the attack, and desquamation of it toward the decline.

Third.—Absence of suppuration.

Fourth.—The special seat of the symptoms, their favorite location being the great toe.

Fifth.—A febrile reaction whose degree of intensity is proportional to the number of joints affected, contrary to what is observed in cases of articular rheumatism.

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[1] Principles of Physic, etc. Vol. ii., p. 753.

[2] I have heard it also put thus: "Screw up the vise as tightly as possible—you have rheumatism; give it another turn, and that is gout."—L. H. H.

And finally, when taking the secondary phenomena into account, the most striking occurrence is the relief felt by the patient after the attack; and this feeling of comparative well-being probably corresponds to the destruction of a certain quantity of uric acid.

Along with this change in the general condition, we ought to describe the alterations occurring subsequently in the local state of affairs. Following the first attack there is, in the vast majority of cases, no restraint whatever to the articular movements; but sometimes you notice prolonged rigidity of the articulation, or an indefinite persistence of the œdematous swelling. These, according to Garrod, are the results of injudicious treatment—such, for example, as the application of leeches to the affected part.

In some cases—happily very exceptional ones—ankylosis occurs at the commencement (Todd, Trousseau, Garrod); and, in other cases, there are premature formations of tophi or chalk-stones.

This, gentlemen, is the commonest type of an attack of regular acute gout. I should undoubtedly have been able to draw a more animated picture of it, if I had not restricted myself to follow an analytical order. But you can readily console yourselves by reading the immortal description left by Sydenham, or the eloquent pages Professor Trousseau has devoted to this subject.

And now, let us see what are the principal deviations which may be presented by the regular type I have just been describing.

Let us first consider the deviations which gout may undergo with regard to the situation of the articular symptoms. Usually, or rather, let us say in the vast majority of cases, the great toe is the part affected—sometimes of one side only, and again of both sides consecutively. Scudamore found that, in 512 cases of gout, the great toe was attacked 373 times at the first fit, either alone or in company with other joints; and of these 373 cases there were 341 in which the symptoms were mono-articular.

It is very clear that, from a diagnostic standpoint, the greatest advantage may be derived from this strange predilection. For in this regard an unmistakable difference subsists between gout and articular rheumatism.

Still, there are cases where the great toe is only affected secondarily, the disease primarily appearing at some other point, as, for example, in the knee. Traumatic causes seem to play a very important part here, a blow or a fall predisposing (as in rheumatism) the injured articulation towards becoming the seat of gout; but we shall return to the subject of etiology farther on.

Exceptional cases do exist where the great toe remains perfectly exempt, both at the first and at the subsequent attacks. Garrod describes cases of this sort, and I have myself noticed a few of a similar character. Gout, then, can be located in the knee or any other joint from the first attack.

Finally, there is a form of the disease that deserves special attention, since it presents the greatest analogy with acute articular rheumatism, at least with respect to the symptomatology: I refer to *acute primitive general gout*, which, from its very first attack, invades several joints at the same time; a great number of the articulations, large and small, may be affected simultaneously. Here the attacks are of longer duration, persisting during two or three weeks, and may even be prolonged over several months. This is the "gout with successive paroxysms" of Professor Trousseau. How many times have not these symptoms been attributed to acute articular rheumatism?

But acute gout presents variations not alone with regard to the seat of its manifestations; but the intensity of the principal symptoms, the pain,

the general reaction, may suffer a strange diminution. This is quite frequently the case with women and debilitated patients, and is the mild, atonic, or asthenic form of acute gout, whose progress is generally unfavorable, and which readily passes into the chronic state.

But, as we have already once observed, gout is essentially a chronic affection, even in its acute form. And hence, it is indispensably necessary to study the attacks taken in connection with one another, to follow them step by step in their repeated returns to the onset, and to describe the characteristics presented by the successive attacks.

Return of the attack.—At the commencement gout seems to accord quite long periods of rest to its subjects: only one attack occurs in two or three years. Later on the symptoms recur annually, and next they appear twice in the year—in the spring and in the fall—a sign that there has already been a change in the usual pathological occurrences, since the first attack generally makes its appearance at the close of winter (Trousseau).

At last the intervening periods grow smaller and smaller, and the attacks return every third or fourth month. Already the chronic state has been entered upon.

You must recollect, in addition, that accidental conditions may intervene or derange this regular progression; a traumatic lesion, phlegmon, or an erysipelas sometimes occurs to accelerate the advance of the disease, or to induce sudden and unexpected returns of it.

Characteristics of the new attacks.—For quite a long period there is no striking modification of the symptoms which collectively mark the attacks. Restricted to one or two joints, the articular inflammation continues to occupy the same locality, while the general symptoms still present the same degree of intensity, the intervals between the attacks being free from all morbid manifestations.

However, as the disease progresses, its character insensibly changes, and it displays a tendency to assume the chronic form. Then it is that the large joints are seen to undergo successive invasion, and that too in almost always the following order: the toes, the instep (*tarsus*), the knees, and then the hands, the wrists, the elbows, and finally in rare cases the shoulder and the hip. Then the patient, struck by the unquestionable analogy, readily imagines that his disease has changed its nature and become transformed into rheumatism.

At the same time the attacks, whose duration is now much greater, possess less intensity, taking on the *subacute* form, and are accompanied by a much more slightly marked febrile reaction. The intervals are much less free from morbid phenomena, and the symptoms of abarticular gout are more and more pronounced: the patient is now troubled more than in the past with dyspepsia, palpitation, and various nervous derangements. In a word, it might be said that the disease, at first concentrated, has ended by becoming diffuse; it gains in extent what it loses in depth. But from this very moment it is chronic gout. Let us, then, study the characteristics of this latter disease.

Chronic Gout.

This form of gout has for its essential characteristic a coincidence with a general depression of the vital forces of the economy, seeming to justify the expressions *atonic* or *asthenic* gout, applied to it by some writers. And

in reality a pronounced feebleness and a tendency to a cachectic condition always do appear—in different degrees of course—when the disease has reached this point. And moreover, intercurrent affections display, in these cases, a most exceptional gravity—influenza, pneumonia and typhus have here an unusual progress, the last-named disease nearly always proving fatal (Schmidtmann, Murchison); and in this respect there might be established an analogy between gout and diabetes—a view which, later on, will be justified by other points of comparison.

We know that here there exists a permanent alteration in the blood and in the urine, which in certain degree explains to us why the intervals between the attacks are full of more or less serious abarticular symptoms, such as palpitations, dyspepsia, and nervous derangements. And it is likewise under the influence of this alteration that the tendency to certain organic visceral affections seems to be generated, the implicated organs being the kidneys, liver, heart, and the vascular system in general.

But chronic gout, though ordinarily succeeding acute gout, may also assume the first-named form from the onset, and in that case it presents slightly different characteristics.

First.—Following acute gout, chronic gout is established very nearly permanently in the articulations, though the local symptoms become less acute and the pains less intense. And lastly, as I have already observed, the upper extremity begins to suffer the invasion of the disease; now it is at this period that you notice articular deformities; it is at this period especially that you observe the production of those tophaceous concretions which are soon to demand our attention in a very special manner.

At the same time, the change which has taken place in the constitutional condition is exhibited during the attack by a reaction which has lost much of its intensity, and during the intervals by more pronounced symptoms arising in the viscera.

Second.—When, on the other hand, gout is chronic from the very commencement, the tophi are seen to form quite early, especially upon the hands. This is the *stationary, primitive form* of gout, in which local symptoms depend almost exclusively upon the presence of deposits of greater or less size.

Here we find grave visceral affections manifesting themselves at an early day in a certain number of patients: Todd has observed albuminuria appearing two years *after* the beginning of the disease; and two years later the patient was seized with epileptiform symptoms and died in coma.

In another patient (reported by Traube), albuminuria existed within one year after the first symptoms of gout, and the body was already covered with tophi.

We shall now consider the clinical characteristics of those concretions which we already know from an anatomical standpoint. And in fact, the tophi, when once formed, possess a certain kind of independent existence, that consequently entitles them to a separate study.

Their diagnostic importance cannot be over-estimated, since they give rise to special deformities belonging exclusively to gout; and, besides this, they are much more frequent than used to be imagined: Scudamore says you meet them ten times out of a hundred, and even that is frequent; but to-day, taking into account the concretions upon the external ear, their existence, according to Garrod, may be affirmed in one-half of all cases.

Their development, which was so excellently described by Moore in 1811, has three stages. Following an attack, in an interval of remission,

and sometimes without any pain,[1] a fluctuating fluid lifts the skin, as has been observed by Cœlius Aurelianus. In the *second* stage, these deposits solidify and assume the form of hard, indolent, more or less rounded masses, increasing with every attack, and even during the interval between them. And finally, in the *third* stage, the skin ulcerates, and there is an escape of chalky material, frequently in considerable quantity, from the aperture thus formed.

When this elimination occurs without phlegmasic action, the cretaceous concretions are left bare ; in England old men with the gout are frequently seen to score their points at cards with the tophi covering their hands, which make a white mark upon the gaming-table like that of chalk. At other times a more or less intense inflammation is lighted up, and we have swelling, redness, then a purplish tint of the skin, and even threatened gangrene ; finally, an opening is made and pus and tophaceous material, the latter composed almost wholly of urate of soda, make their escape.

In some cases this phlegmasia results in the formation of ulcers which are very difficult to heal. The urate of soda infiltrating the mesh-work of the cellular tissue renders it difficult to cleanse the sore, while the cicatrices also have a tendency to reopen.

Sometimes the articulations themselves may be encroached on, but it is worthy of remark that this occurrence portends no serious danger to the patient.

The flow of this material often leads to local or general relief ; and Garrod has even stated that when attempts have been made to repress this action by the application of astringents, articular pains sometimes reappeared either at the point where the ulceration occurred, or at some more distant spot. When it has reached its last phase, gout develops a cachexia, whose principal elements are marked anæmia, extreme muscular weakness, especially in the lower limbs, and intense depression of the nervous system; the patients are incapable of undergoing the least fatigue, and the slightest noise becomes intolerable.

And here, gentlemen, we finish the clinical history of regular gout. This form of gout is pre-eminently characterized, as you are yourselves convinced, by a marked and frequently exclusive predilection for the articulations. And this is the type which you will meet with oftenest in your practice, and which will be the easiest for you to recognize.

We cannot say as much for the anomalous, the irregular, the masked forms of gout ; for they sometimes assume the guise of the most different affections, those farthest removed from the gouty diathesis, and the physician who has failed to become specially versed in the diagnosis of cases of this kind, is very liable to commit errors which may involve the most deplorable consequences. Here, then, is a subject deserving our prolonged attention, and all the more so since its study will enable us to appreciate one of the most essential points of difference between the spirit of medicine of ancient days and that of contemporary science. We shall consider this subject in our next lecture.

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[1] I have recorded a case of gout, where the patient could never suffer a pain, even a transient one, upon any point whatever, without the immediate formation of a tophus at that spot.—Note by the Editor.

LECTURE VII.

SYMPTOMATOLOGY OF VISCERAL GOUT.

Summary.—Predilection of the Ancients for the Study of Pathological Metamorphosis—Importance of Masked Gout in this Respect—Scepticism of the Moderns—Definition of Visceral Gout—*Functional* Derangements: *Organic* Lesions—Masked, Misplaced, Retrocedent Gout—Can Visceral Gout exist Independently of all Articular Disease?

Diseases of the Digestive Canal—Spasm of the Œsophagus—Dyspepsia, Cardialgia, Gouty Gastritis—Hepatic Evidences of Gout—The Circulatory Apparatus: Lesions of the Heart and Blood-vessels—Sudden Death—Cerebral Manifestations of Gout—Its Influences upon Diseases of the Spinal Cord not yet Demonstrated—The Respiratory System: Gouty Asthma—The Urinary Passages: Frequent Disease of these in Gout—Functional Derangements of the Kidney—Gouty Nephritis—Indication of a few other Abarticular Diseases which accompany Gout.

GENTLEMEN:—As I announced at the close of my last lecture, we shall now commence the study of visceral gout. This subject, as I have already remarked to you, deeply engaged our predecessors, and it will be an easy matter for you to comprehend the reason therefor.

Accustomed to *collective* views, and taking but little care to minutely analyze clinical facts, the physicians of by-gone centuries always had a marked predilection for the study of the metamorphoses which diseases of long duration underwent. From Galen to Roderic a Castro, who published in the seventeenth century a curious book bearing the singular title *Quæ ex quibus*, and from that time to Lorry, a great number of writers have undertaken to describe the transformation of diseases (*mutationes morborum*). Gout occupies a prominent position in these works, and is more than once invoked when they desire to prove that a disease may assume the most diverse forms and still not lose its identity.

It is a generally recognized fact, to-day, that our predecessors exaggerated the number and the frequency of the transfigurations which pathological conditions might undergo. Now, in our times, this study, which of old was so flourishing, has fallen somewhat into disuse, or, let us rather say, it is regarded from an entirely different standpoint.

But, particularly concerning masked gout, we must acknowledge that the ancients saw it everywhere, even where it did not exist. We cannot, however, range ourselves by the side of those physicians who, under the influence of the too radical reaction of to-day, have even gone so far as to deny the existence of this form of gout; such scepticism is too arbitrary. Visceral gout is a relatively infrequent disease; but it does exist—at least, we hope to prove to you that it does. The only question is to ascertain, by careful analysis, of what it consists, and what limits are properly to be assigned it.

Situated as we now are, I do not deem it necessary to vindicate the importance of this subject in your eyes—it is one which recommends itself to your attention from a double standpoint; for, in regard to general

pathology, the obscure and controverted question of metastasis and retrocession is most intimately connected with this study; and as to special pathology, the history of the visceral manifestations enables us to appreciate simultaneously the great resemblances uniting gout and rheumatism, and also those differences which separate the two.

But, before leaving the domain of the clinic to commence the description of particular phenomena, it seems indispensably necessary to determine, with as much precision as possible, the meaning of the expression *visceral gout*.

This term must not be indiscriminately applied to all the diseases which may occur in a patient with the gout; these diseases—some purely accidental, and others having but a very remote relationship to gout—are modified, it is true, by the nature of the soil in which they develop; but they cannot be reckoned in with the direct consequences of this affection.

We shall reserve the name *visceral gout*, then, for those morbid phenomena that may develop in our internal organs under the immediate influence of the gouty diathesis; and in this pathological series we shall distinguish two natural groups: the first includes *functional* derangements arising from that general condition; and the second embraces the *organic* lesions which may be evolved under its influence.

To the first of these two groups are especially applied the expressions masked, misplaced, and retrocedent gout—expressions encountered at every step in writers on the subject; while, to give a homogeneous character to this order of morbid phenomena, we ought not to assign to it any but those visceral affections which, in all essential points, are analogous to the articular lesions of gout, and play a similar rôle in the pathological drama, if we except the locality they choose for their seat.

In order that you may better grasp my idea, allow me to cite a few examples. An individual who, for a long time, has been a dyspeptic, suddenly suffers an attack of gout, and is cured of his dyspepsia—at least, he is so to all appearances; but, once the articular symptoms are quieted, the stomach is again troubled as of old. This, gentlemen, is an indisputable case of visceral gout, where the stomach seems to take the place of the articulations in the series of morbid manifestations, and is called upon, after a fashion, to suffer in their stead. In like manner, it is known that a patient subject to epileptiform convulsions may become gouty, and that a cure of the nervous symptoms may be the result. Garrod has collected several observations of this nature.

In cases of this kind, the visceral affection seems to consist in a purely dynamic derangement; or, at best, only a superficial change in the tissues exists. And, besides, you readily understand how the gravity of these manifestations is subordinate to the locality they occupy.

Very rarely, however, do you notice here those crystalline deposits of urate of soda, which, in the cartilages and in fibrous tissue, commemorate, as it were, the history of previous attacks. And besides, the anatomical elements may be impregnated with urate of soda—as I have previously described—and still not present crystalline deposits. These affections are essentially mobile in their nature; they appear and disappear suddenly; they may coexist with, precede, or follow articular symptoms; but, in the majority of cases, they are seen to alternate with them. In those cases where the visceral trouble precedes articular gout, and, for a longer or shorter time, constitutes the only evidence of the diathesis, the name *masked* (larval, *larvée*) *gout* is applied to it. When, on the other hand, it follows symptoms occurring in the joints, it is called *retrocedent* gout, pro-

vided, always, the metastasis has been brought about by the evident intervention of some external cause—as, for example, cold. Finally, we say that *gout has retroceded of itself* when the symptoms change their locality *spontaneously*.

And here we have to solve one of the most difficult problems in the study we have undertaken. Can symptoms of visceral gout be present in a patient whose joints never have been and never will be diseased? In other words: can larval, or masked gout exist independent of articular gout? It seems probable, at least; and yet how many difficulties crowd around its demonstration!

Let us observe, however, that these phenomena may be present in one born of gouty parents, and who from the very outset is thus evidently predisposed to gout. Here is the first presumption in favor of the hypothesis we have undertaken to defend. In the second place, visceral gout appears in one of the forms which it ordinarily assumes, when it coexists with articular gout. In the third place, there are cases in which implication of the joints shows itself as a rudimentary condition, evinced by painful *twinges*. And lastly, the uric acid diathesis, characterized by the group of phenomena which we have previously described, may give such an imprint of authenticity to these visceral manifestations of gout, that it can scarcely be called in question, when once the presence of *uric acid in the blood* has been established.

There now remains the second group, that of *organic* lesions, which in the long run almost always succeed affections of the first kind, and have the same situation. Still, we are far from admitting that an absolute distinction exists between these two orders of occurrences, while it is very reasonable to believe that the functional derangements are but the initial stage of those textural changes which give rise to permanent disease.

It is imperative, gentlemen, that we should reduce the very complicated nomenclature of gouty affections to the simplest terms; and to impress these ideas yet more clearly upon your minds, we shall present a *résumé* in the following table:

VISCERAL GOUT..... { FUNCTIONAL..... { *Masked, larval* (preceding). *Retrocedent* (following). ANATOMICAL, *with permanent lesions.*

I.—We shall now study in succession the two forms of visceral gout which we have distinguished, in each organ and in each system that can become its seat.

We shall begin with the digestive tract, for it is here that affections of this character are especially developed; and it can be asserted with reason that "*gout is to the stomach what rheumatism is to the heart.*"[1]

A.—We shall only say a word concerning quite a rare disease of the œsophagus, described by Garrod: this is a spasmodic constriction of the tube, obstructing the passage of the alimentary bolus. An attack of gout is followed by disappearance of this condition.

B.—Next in order follows the discussion of gastric gout, a subject we have already slightly touched upon when speaking of the chronic dyspepsia of gouty patients, as well of the nervous symptoms which accompanied it.

Masked (larval) gout of the stomach precedes the attacks or fits, and

[1] Ball: Thèse pour le Concours de l'Agrégation, p. 158.

may even develop before any articular disease: in a very large number of subjects the gastric derangements undergo marked improvements as soon as the joints become implicated.

In one patient—a case which I personally observed—the digestive troubles were present before the first attack of gout; the diagnosis was made from the existence of a tophaceous concretion on the external ear, and the subsequent progress of the symptoms completely substantiated my statement.

In another case there had been *only a single attack* of articular gout. Later on intense dyspepsia occurred, and, after having invoked the aid of the regular practitioner to no purpose, the patient determined to summon a homœopathist. An unexpected success crowned the method of treatment, and he was already congratulating himself for having had recourse to the "new medicine," when suddenly he was attacked with gout in the foot, thus affording an explanation of the miraculous cure. Here was a visceral manifestation of the diathesis.

Gout *retroceded* to the stomach (*gout in the stomach*) differs from masked gout in the gravity it may assume; here it is that we often have the opportunity of observing those ominous symptoms which sometimes terminate in death.

Gout, it is claimed, may retrocede of itself; this is the spontaneous metastasis of Guilbert. But much more frequently this displacement is induced by the intervention of a direct cause. The regular progress of the disease is broken by some violent emotion, by an attack of indigestion, or by ill-timed treatment: the patient, urged by his unendurable suffering is imprudent enough to plunge the affected member into ice-water (Lynch, Parry), or to administer to himself some special remedy, as, for example, colchicum (Trousseau, Potton de Lyon). Then, as if by magic, the pain and swelling of the joints subside, he already felicitates himself upon the treatment he has resorted to, when the formidable phenomena of gout in the stomach are suddenly seen to break forth.

It is expedient to distinguish, at this point, with Budd and Scudamore, two symptomatic varieties. In the *first* case, the disease announces itself in a *cardialgic* or *spasmodic* form: there is intense pain relieved by pressure, together with a feeling of cramps in the epigastric region; at the same time marked distention of the stomach occurs, accompanied by attacks of vomiting which are frequently uncontrollable, and a more or less grave general condition. Then algidity and cold sweats supervene, the pulse becomes small, frequent and irregular, and a tendency to syncope is exhibited. In cases like these, according to Cullen, stimulation especially is to be employed, and alcohol in large doses is tolerated.

In the *second* case the disease assumes an inflammatory form. There is intense epigastric pain, especially on pressure, and repeated attacks of vomiting occur, the matters being almost black, or sanguinolent; there is a more or less intense febrile movement, and following these phenomena comes a condition of general prostration. Stimulation is not to be employed here, bloodletting being the treatment commended in these cases.

Where the general health is very poor, all this train of symptoms suddenly disappears, either under the influence of the proper kind of medication, or spontaneously with the gouty inflammation returning to the great toe. There is a general belief in the influence exerted by stimulating applications to the joint first affected, to recall to that spot a gout which seems to have been displaced; but there are scarcely any well-authenticated facts to be found in books corroborating the usefulness of this mode of treatment.

But science has recorded eight or ten instances where these symptoms have ended in death.

At the post-mortem examinations which have from time to time been made, a thickening of the submucous cellular-tissue was discovered; the mucous membrane of the stomach was œdematous and covered with hemorrhagic erosions, and the cavity of the organ in *some cases* contained a black fluid. These lesions, collectively considered, seem to indicate that a change had already taken place at some distant period, notwithstanding the sudden occurrence of the disease.

But, fortunately, these terrible cases are quite rare. Scudamore mentions only two or three of them, and Garrod and Brinton never met with one. Quite recently Budd and Dittrich published two such cases.

But, in a much milder form, gout in the stomach is quite a common disease; it is chiefly met with in cases of asthenia and gouty cachexia, and in individuals who have abused specifics and the application of leeches and cold.

It may be asked, however, if the existence of this disease has not been too readily admitted in a certain number of patients. It is unquestionably very easy for errors in diagnosis to be made here; for hepatic or renal colic, the digestive derangements accompanying albuminuria, and perhaps even poisoning from certain remedies (from colchicum in particular)—all these have more than once simulated gout in the stomach. A simple attack of indigestion, abruptly occurring in a gouty patient may possibly be mistaken for an attack of retrocedent gout, on account of the gravity often assumed by the symptoms, because of the special predisposition induced by the uric acid diathesis. Thus, Watson remarks that we should say "*pork* in the stomach, instead of *gout* in the stomach." This scepticism has even gone so far as to almost absolutely deny the existence of gout in the stomach, and Brinton, after a long discussion of this question, finally concludes that a little gastric irritability may *possibly* exist in gouty subjects, but that anything beyond this must be regarded simply as a coincidence.

We profess the opposite opinion in this respect, and after assigning a large share to errors in diagnosis, we believe that the various diseases which may be summoned to do duty in this regard are yet far from accounting for all the phenomena. We have previously seen that physiological experimentation succeeded in producing in animals occurrences that were analogous to those of the gouty diathesis; and in such cases we know that the gastric juices and the stomach-follicles become charged with urate of soda. While not pretending to call upon identical conditions to account for the phenomena of gout in the stomach, we think that superficial lesions can very readily be developed in the digestive apparatus under the influence of retrocession; and this view is perfectly *confirmed* by the results of autopsies made upon gouty patients who for a long period of time have suffered from their digestive organs. In such cases as these Todd has often discovered an enormous dilatation of the stomach, and Brinton has himself verified these results by personal observations; this is the paralyzed and enfeebled condition of the stomach which Scudamore long since described as existing in chronic cases of this disease.

And besides, it is probable that, after a time, permanent lesions are developed in subjects liable to these apparently functional manifestations; and the fatal cases to which we have just alluded seem to afford the proof of it.

C.—Corresponding to the two forms of gout in the stomach, above de-

scribed, are two forms of intestinal dyspepsia: the first is characterized by attacks of spasmodic colic, and the second is a true enteritis. These phenomena may exist separately, or be joined to the various derangements of which the stomach may become the seat.

II.—"*The liver is rarely healthy in gout*" says Scudamore, and daily observation tends to demonstrate the truth of this opinion.

There are, undoubtedly, diseases of the liver associated with gouty dyspepsia, and also transient enlargements of this organ which precede the attack, such as were observed by Scudamore, Galtier Boissière, and Martin-Magron.

But we are not yet absolutely certain whether there are permanent diseases of the liver which are the result of the gouty diathesis. Scudamore thinks that, in the end, the spleno-hepatic system feels the influence of gout, and becomes the seat of permanent disease. Besides, from the labors of modern physiologists, we know that the liver and spleen are probably the organs in which uric acid is formed.

Still the anatomical character of this visceral affection (if it exist), remains as yet unknown to us; and the lesions in the liver met with in gouty subjects are almost always the results of alcoholismus.

Biliary gravel, however, is sometimes coincident with the uric acid diathesis and gout (Prout, Budd, Wunderlich, Willemin), and now and then you may meet with uric acid calculi in the gall-bladder, according to Stöckhardt, Faber, and Frerichs. Still, the patients in whom the concretions were discovered might have been gouty.

III.—The influence of gout upon diseases of the heart cannot be controverted. But we are not concerned here, as in rheumatism, with endocarditis, pericarditis, or valvular lesions; these conditions, when occurring in the gouty, seem to be caused especially by Bright's disease and alcoholismus.

The dominant condition here is a fatty degeneration of the muscular tissue of the heart. Stokes, Quain, Gairdner, and Garrod all unite in affirming the existence of this state of affairs.

In the beginning this affection is present only in a slight degree, and merely manifests itself by functional derangements—palpitation, dyspnœa, and feebleness and irregularity of the pulse (Hervez de Chégoin). Gouty retrocession is quite infrequent (Scudamore and Garrod), though there are a few cases of it, and death has even resulted from cardiac implication. In these cases, however, the lesions we are about to study had already been developed.

In the second stage, we find fatty degeneration of the heart. The symptoms of this disease are always the same, whatever be the cause of it (Stokes, Garrod). They simulate functional derangement, and, the physical signs being only slightly marked, the diagnosis is arrived at chiefly by a method of exclusion.

The cardiac impulse is feeble, almost absent; the first sound is dull, and sometimes there is a murmur resulting from fatty degeneration of the tenser muscles of the valves. The precordial dulness is frequently increased; the pulse is soft, compressible, intermittent, and sometimes exceedingly slow, especially during an attack or paroxysm (twenty to thirty pulsations); and finally, some have described the *arcus senilis* (Canton) as existing in those individuals whose cardiac muscular tissue has undergone this alteration.

The rational symptoms are also quite capable of deceiving the observer.

The attacks develop by paroxysms; there are violent palpitation, dyspnœa, and a tendency to syncope; and cerebral symptoms are seen to occur, assuming the guise of an apoplexy, although no intracranial hemorrhage has taken place (Law, Stokes). Sharp pains develop about the precordial region and shoot down the arm, thus simulating angina pectoris, which is itself frequently regarded as a disease of gouty origin.

Finally, sudden death is a very frequent occurrence in these cases: thus, in 83 cases of fatty degeneration collected by Quain, death took place unexpectedly in 54, viz.: 28 times from rupture of the heart and 26 from syncope. In many of these observations the subjects were gouty patients.

It is evident, then, that a goodly number of cases where death was attributed to *gout in*, or *retroceded to the heart*, were but cases of fatty degeneration of the organ. Quain and Gairdner witnessed death produced without rupture under these circumstances, while death from rupture was observed by Cheyne and Latham. The fatal termination has often occurred during an attack of gout, this latter condition seeming to act here as an incentive to the cardiac crisis.

Lastly, let us note that an atheromatous condition of the arteries, very frequently coexisting with these cardiac lesions, may give rise to cerebral hemorrhages, in which case true, and not false apoplexy, is observed.

IV.—Our ideas concerning the connection between multiple arthropathies and diseases of the nervous system have undergone a definite change—most occurrences of this kind used to be attributed to gout; but to-day, rheumatism, through the researches of modern investigators, has been accorded the first place. Nevertheless, gout still retains its share in it, though it is interesting to notice that the two diseases have, in this respect, a parallel progression, and that all forms of cerebral rheumatism are found again in gout.

Thus, the rheumatismal headaches described by Van Swieten, and more recently studied by Gubler, have their counterpart in the gouty cephalalgiæ which have so long been known, and which, in later times, have been so carefully described by Lynch,[1] Garrod, and Trousseau. Acute delirium, or the meningitic form of cerebral rheumatism, occurs also in gouty patients, according to Scudamore. Rheumatismal apoplexy, or the apoplectic variety of cerebral rheumatism, appears in the form of stupor in gout, according to Lynch and Professor Trousseau.

The convulsions manifesting themselves during the course of encephalic rheumatism are likewise present in gout; though in rheumatism they especially assume the form of chorea, while in gout they are rather epileptiform in character, as Van Swieten, Todd, and Garrod have observed.

And, finally, we know the existence of a rheumatismal lunacy, which has been studied by Burrows, Griesinger, and Mesnet; and the same is true for gout, according to Garrod. Lunacy, however, in the latter disease is rare, at least in France—Baillarger, whose experience is authoritative in these matters, having informed us that he has never met with a single instance of it.

Just here let me remark, while pointing out a difference: that *aphasia* which is never present in rheumatism, (if we except cardiac diseases and secondary embolism), is met with, on the other hand, in gout. It must be

[1] We recommend to our readers the memoir of Lynch as deserving of attention: Some Remarks on the Metastasis of Diseased Action to the Brain in Gout, etc. Dublin Quarterly Journal, p. 276. 1856.

confessed, too that encephalic derangements are, in general, less grave in gout than in rheumatism, that their alternation with articular phenomena is more marked, that retrocession is more palpable, and finally, that although there often exists a masked (larval) cerebral gout, this is very rarely the case in rheumatism.

Cerebral symptoms of gout must not be confounded with *delirium tremens* occurring at the moment of the attack (Marcet), with the delirium which appears in acute intercurrent affections, or finally, with the symptoms which dyspepsia, cardiac lesions, and uræmia develop on the part of the nervous system with much greater frequency in gout than in rheumatism. In this regard, prolonged observation, and an attentive study of the patient during a long period of time are the only means by which all error can be avoided.

V.—The influence of gout upon diseases of the spinal cord is still a mooted question. Todd and Garrod speak of the appearance of slight symptoms, a sort of *paralysis* alternating with the attacks; but you must not confound a lesion of the spinal cord with that muscular feebleness which follows intense paroxysms of articular gout, and which may even simulate actual paraplegia.

True, Graves has reported a case where, upon an autopsy being made, a hardened and shrivelled condition of the cord was discovered; but this example does not appear to us to be quite satisfactory. Besides, it must not be forgotten that if there do exist medullary diseases associated with gout and rheumatism, which is as yet unproven,[1] there are, undoubtedly, strongly marked articular diseases which may develop subsequently to lesions of the spinal cord, even if these be of traumatic origin.[2]

VI.—The respiratory apparatus may likewise become the seat of certain gouty manifestations, and these we shall rapidly pass in review:

First.—Gouty asthma.—Among thoracic affections deserving of this appellation, there is one which corresponds to that variety of asthma called *nervous;* the lungs are perfectly free during the interval between the attacks, and there is an evident alternation between the thoracic and articular symptoms.

Dr. Vigla has reported a very interesting example of this kind to the "Medical Society of the Hospitals."

There is, however, a *second* form of gouty asthma, arising from permanent lesions and which especially co-exists with emphysema; indeed, you recognize here alternate exacerbations and remissions corresponding to the disappearance and return of articular symptoms. These cases are quite rare: Patissier saw only 2 out of 80 cases; Garrod, 1 out of 40; and Hyde-Salter, to whom we owe a treatise on asthma, also reports but a single case of it.

Second.—Some of the ancient writers describe a gouty pleurisy. This was probably a simple gouty pleurodynia, as we shall see farther on.

Third.—Is there a gouty pneumonia? It has been mentioned by some observers, but authenticated cases are yet wanting. Scudamore has twice seen gout manifest itself after the disappearance of a pneumonia. The

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[1] To-day it certainly has been demonstrated that there is a rheumatismal *myelo-meningitis.* Two observations at *post-mortems* quite recently made in Professor Béhier's service furnish irrefragable proof on this point.

[2] Ball: Thèse de Concours pour l'Agrégation, p. 87. 1866.

question arises whether this be not a fortuitous coincidence. And, according to the same authority, these two diseases may always coexist without exercising any influence over each other. We shall soon recur to this point.

VII.—Diseases of the urinary passages are quite frequent in gout, and at a certain phase of the disease they become almost the rule. They are, on the contrary, rare in the various forms of chronic articular rheumatism. And herein we discover a characteristic distinction of a not uninteresting nature.

It is necessary, however, to eliminate from the domain of visceral gout, in accordance with the rules which we have hitherto followed, everything pertaining to calculi and uric gravel, whether renal or vesical. It is true that these occurrences are frequent among the gouty, but they do not exclusively belong to the diathesis.

Still, there are urinary diseases traceable directly to gout, and these we shall now describe.

A.—The kidneys may suffer a transient functional derangement, presenting striking analogies with articular gout. This occurs in less advanced periods of the disease, having for its symptoms a sharp but transitory pain, manifestly alternating with articular gout, and which may have its seat in either kidney, accompanied by an albuminuria of short duration. It may be that during the entire course of this complication there is no emission of gravel.

This is not an exceptional manifestation of gout; Garrod has observed several examples of it, and I have myself, with Dr. Clin, seen one case. This patient was a physician, and therefore could give descriptions of the phenomena he had experienced, worthy of reliance.

B.—Permanent disease of the kidneys becomes almost the rule in chronic gout; in this case there is an *albuminous nephritis*, presenting anatomical characteristics which leave no doubt as to their origin, namely, infarctions of urate of soda in the renal parenchyma. We have already described these changes when speaking of pathological anatomy.

Once established, gouty albuminous nephritis differs but slightly, in respect to symptomatology, from ordinary Bright's disease. The urine is generally found clear and with but little color, containing a varying, although almost always a small quantity of albumen, only a trifling amount of urea and salt, and exhibiting under the microscope fibrinous cylinders, covered with epithelial cells or loaded with granulations. There may be œdema of the face and lower limbs, though this symptom is very frequently absent. As in Bright's disease, so here we find dyspepsia and diarrhœa; but, as we have already hinted, the progress of the malady is slower and its prognosis less grave than in albuminous nephritis properly so called.

Nevertheless, uræmia has sometimes been observed to occur in the course of this disease; Basham, Todd, Dechamps (*of Bordeaux*), and other investigators have recorded several examples of it. Some years ago I called attention to this point myself, and Fournier has shown the importance of it in his remarkable *thèse de concours* (prize thesis) upon uræmia.

C.—A *vesical gout* has been described by various authors: Scudamore speaks of it, and Todd has endeavored to set forth its characteristics;[1] indeed, a large number of those cases which the English designate by the name of *irritable bladder* ought to be ascribed to this.

[1] Todd: Clinical Lectures on Certain Diseases of the Urinary Organs, p. 359. London, 1857.

At the commencement it is merely a question of a transient affection—precisely as we saw occurring in the case of the kidneys—merely a dynamic derangement, characterized by sudden and violent pain in the bladder, by tenesmus, and by a flow of blood and muco-pus from the urethra, even in the absence of all calculous complications (Todd). These phenomena may alternate with the articular disease.

But at a more advanced stage there is a permanent lesion, with catarrh of the bladder and other phenomena of that kind. Professor Laugier was good enough to communicate a case to me proving the reality of vesical gout, independent of all gravel complication.

D.—A gouty *urethritis*, with escape of pus from the urethra, has finally been mentioned; but have not the writers (Scudamore in particular) allowed themselves to be imposed upon? Perhaps it was a case of blennorrhagic arthritis—such, at least, is the interpretation which may be given to some of Scudamore's observations.

VIII.—*Abarticular—non-visceral—gout.*—Independent of the internal diseases that may be developed during the course of gout, there are other manifestations of the same disease, which, though not invading internal organs, are yet localized elsewhere than in the joints: they are seen occupying the muscles, the nerves, the skin, and some other portions of the economy.

A.—In the first group we shall place phenomena of this kind which belong to ligaments, tendons, and fibrous tissue in general.

We have previously established the fact that, when the articular (diarthrodial) cartilages have been saturated with urate of soda, the ligaments and tendons are then observed to become impregnated with that substance. The symptoms of this condition of affairs are generally merged into those of articular gout, though they may sometimes play a separate and independent rôle. It is well known, for example, that the fibrous tissue in front of the patella (*pre-rotular*) may be the seat of pains that are strikingly analogous to those of acute gout; this is Rayer's *prerotulian gout*. It may be asked, then, if gout can exist in a larval or masked form in tendons and ligaments; can it precede, or can it wholly take the place of articular gout? This is still an obscure point, and one demanding new investigation.

B.—*Muscles.*—Gouty subjects frequently experience painful cramps in the muscles of the extremities during an attack of the malady. They suffer very often from lumbago, and may even feel very intense pain at certain points in the chest-wall; these are probably in the intercostal muscles or in the fibrous tissue of the thoracic parietes (*gouty pleurodynia*). The patients very often designate these sensations by the name of *rheumatic pains*, although in reality they belong to gout.

C.—*Nerves.*—Pain along the course of nerve-trunks, particularly of the sciatic and trifacial, is also known to be present during the course of gout. Its characteristics are an abrupt appearance and a similar departure, alternating with articular symptoms.

D.—*Cutaneous affections.*—Brodie and Civiale long since observed an evident relationship existing between psoriasis, gravel, and uric acid calculi. It is certain that psoriasis may often be met with in those belonging to a gouty family, and that it may coexist with gout in one and the same individual. This fact has been placed completely out of question by the observations of Holland, Garrod, and Rayer. Eczema has likewise been seen to alternate with the most characteristic attacks of gout.

Such, for the time being, are the only cutaneous diseases whose correla-

tion with gout is authentically established. But, under the title *arthritides*, Bazin and his school have collected a great number of different exanthemata arising from rheumatism and gout, two diseases which they blend into one—*arthritis*. It must be confessed that the observations they present in support of these opinions are not by any means all that could be desired, and that gout is here left completely in the background. The cases which Bazin collected belong exclusively to rheumatism.

E.—*Ocular affections.*—Morgagni has already described the conjunctivitis sometimes occurring in the course of the first attack of gout, and concurrent observations have been made by several authors succeeding him, but of all the ocular diseases associated with gout, the one which deserves, in the highest degree, to be joined to the diathesis is certainly *iritis*. Lawrence and Wardrop report cases where the alternation of iritis and distinctly marked gouty attacks could not be called into question; and Professor Laugier communicated a case to me where this phenomenon was perfectly exhibited. And it is very remarkable indeed to see the iris thus affected in gout, the more so since we know that in articular rheumatism and in *subacute*, *nodular* and *blennorrhagic* (gonorrhœal) rheumatism especially, the same symptoms are induced. Lastly, Garrod, in a recent article [1] has described a gouty affection of the eye, to which attention had not yet been called—we mean inflammation of the sclerotic, with whitish deposits of urate of soda upon the surface of this structure.

F.—*Diseases of the auditory apparatus.*—We have already dwelt so long upon tophaceous concretions of the external ear, that there is no necessity of again returning to that subject; and we have likewise pointed out the alterations in the ossicles of the ear. Now, it seems certain to us that patients suffering from chronic gout are liable to become deaf; and it would be interesting to be able to class this new species of deafness along with the lesions we have just been describing, but as we know nothing positively in this connection, new investigations are necessary to fix our ideas upon this point.

Here we shall end the history of abarticular and of visceral gout, and in our next lecture we shall take up some diseases that offer a certain degree of relationship to gout.

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[1] Reynolds: System of Medicine. Art. Gout, by A. B. Garrod.

LECTURE VIII.

CONCOMITANT DISEASES OF GOUT.

Summary.—Conditions seemingly Allied to the Gouty Diathesis—Uric Anthrax—Grave Phlegmonous Inflammations and Erysipelas—Dry Gangrene—Intercurrent Diseases in Gout—Its Affinity with Diabetes—Greater or Less Frequency of this Relation—Diabetes, Obesity and Gout frequently met with, if not in the Same Individual, at least in Different Members of the Same Family—Observations in Support of this—Practical Results—Gravel—Urinary Concretions—Uric Acid—Oxalic Acid—Formation of a Uric Acid Sediment not always Proof of Augmentation in the Secretion of this Acid—Gravel sometimes Associated with the Presence of an Excess of Uric Acid in the Blood—Real or Supposed Correlation of Gout, Scrofula, and Phthisis; of Gout and Cancer; of Gout and Rheumatism.

GENTLEMEN :—At that portion of our course where I thought it expedient to give the history of ab-articular affections, it would have been premature in me to dwell upon certain occurrences which likewise seem to be more or less directly associated with the gouty diathesis. The time has now arrived when it is desirable to say a few words concerning them.

I.—A peculiar predisposition to dangerous phlegmonous inflammation (*p. de mauvaise nature*) and to sphacelus (gangrene) had long ago been observed in diseases which induce a profound change in the constitution of the blood (*crasis*).

Albuminous nephritis is an example of this kind: gangrenous erysipelas or diffuse phlegmon in regions either infiltrated spontaneously, or as a result of incision or puncture, is frequently seen to occur in patients suffering from albuminuria. On this account, punctures and scarifications, which in other dropsies are a means of relief, are in this case expressly contra-indicated (Rayer).

Diabetes offers us a second example of this unfortunate predisposition. English physicians had long since noticed that anthrax, dry gangrene and diffuse phlegmon supervened with the utmost readiness in diabetic subjects, and Marchal (*de Calvi*), unaware of the labors of his predecessors, had the merit of calling attention to this point, which, prior to his time, had scarcely been studied in France.

Now we shall give an account of an analogous series of phenomena occurring in the uric acid diathesis and in confirmed gout.

This pathological coincidence, which already had been adverted to or described by Morgagni, Thompson, Schonlein, Ure, Carmichael, and Prout, has in our times been demonstrated through the labors of Marchal (*de Calvi*), who has concisely divided diabetic symptoms from those which arise from gout—a work which has been accomplished by none of his predecessors.[1]

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[1] Morgagni: De sedib. et causis morborum. lib. iv., epist. iv., § 24 et seq. A. R. Thompson: Histology of a Case of Dry Gangrene, in Medic-Chirurg. Transactions, Vol. xiii., p. 178. 1827. Schonlein: Patholog. und Therapie, p. 248. Bd. iii. The author

Cases of this kind, which arise from the uric acid diathesis, may be divided into three principal classes:

First.—The *uric acid anthrax* is considered as accidental by Garrod and Trousseau when it occurs *before* the appearance of gout; but, when present during the course of the disease, a symptom of this kind seems to arise from the gouty diathesis. Ledwich and Marchal (*de Calvi*) have reported several such cases.[1]

Second.—Prout has described the dangerous *low phlegmon* and *erysipelas* that may occur in gouty patients.[2] Besides, the operation for cataract is known to succeed poorly in these individuals, since the eye almost always becomes inflamed.[3] We may associate with these phenomena an instance of suppuration of the eye-ball in a case of chronic gout,[4] and one of faulty consolidation of fractures. In a case in which there was a fracture of the external malleolus, an attack of gout was developed, and then the fragments separated, ulceration setting in and the bone became denuded; but when the attack ended, everything was restored to its proper form.[5]

Third.—*Dry gangrene*, described by Carmichael, Rayer, and Marchal (*de Calvi*) occurs especially in those debilitated patients who are attacked with the chronic form of gout and in whom tophi or chalk-stones exist.[6]

II.—Let us now direct our attention to the intercurrent diseases of gout.

We are no longer dealing with affections subordinate to the gouty diathesis, but with actual complications that occur during the course of the malady. In what way are intercurrent diseases modified by gout? In this regard we may institute a comparison between Bright's disease, diabetes, and the present subject of our consideration. Indeed, gout has consequences resulting from traumatism such as we have just been describing to you, and thus we observe in that fact one point of union between these three conditions.

According to Prout, phlegmasiæ frequently assume an adynamic form in patients who are gouty, especially if they are also strumous or obese: in this manner, he says, the greater number of them die.[7] And here is an-

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attributes to arterial ossification that gangrene of the extremities which, according to him, is so frequently observed in gout. Al. Ure: Researches on Gout. Medic. Times, Vol. iii., p. 145. 1845. Carmichael: Dublin Quart. Journ., Vol. ii., p. 283. 1846. Prout: Stomach and Renal Diseases, p. 211. 1848. Marchal (*de Calvi*): Recherches sur les Accidents Diabétiques. Paris, 1864.

[1] Trousseau: Clin. Méd., Vol. iii. Garrod: On Gout. Second edition. London, p. 285. 1863. Ledwich: Dublin Med. Journ., p. 43. Vol. xxv. Marchal (*de Calvi*): Loc. cit., pp. 38, 283.

[2] Loc. cit., p. 213.

[3] W. Budd: Library of Medicine, Vol. v., p. 213.

[4] Critchett: Med. Times, Vol. i, p. 62. 1853.

[5] O'Reilly: American Medical Times, p. 39.

[6] Carmichael: Loc. cit. Rayer: Communication Orale. Marchal: Loc. cit.

[7] As this passage has frequently been quoted in the course of the lectures, I shall give it *verbatim*:

"It is not rare to meet with this coincidence (uric acid gravel and diabetes) in obese individuals of mature years, whose temperament is both gouty and strumous, and it ought always to cause the physician great solicitude. I have ascertained that such patients succumb, in general, to some visceral inflammation as sudden as it is violent, and which rapidly takes on the adynamic type. Besides, they seem exposed to grave forms of erysipelas and to diffuse phlegmon. Finally, in most of the cases which I have seen terminate fatally, either from spontaneous diffuse inflammation, or from that induced by simple puncture, the urine had from time to time contained sugar, and had deposited uric acid concretions and gravel."—Prout: Stomach and Renal Diseases, p. 211. 1848.

other point by which we can approximate it to albuminuria and diabetes. Typhus is an exceptionally grave affection in gouty subjects; according to Schmidtmann and Murchison, it is always fatal.[1] And syphilis, according to Wells, is very serious when occurring along with gout; it is very apt to take on a scorbutic character.[2]

These occurrences, peculiar to the gouty cachexia, are to be explained, according to Garrod, by the impermeability of the kidneys.[3] A rapid tissue-metamorphosis sometimes demands an enormous elimination, and this cannot take place when renal excretion is insufficient. But it is probable that this is a more complex problem than the English writer supposed; and the crasis of the blood ought to play a great part therein. We can at least say that, under the influence of such a diathesis, the mechanical, physical and chemical phenomena of life ought to proceed with much greater difficulty than in the normal state of affairs.

When, however, no cachexia is present, the termination of these diseases is far from being so fatal, and things go on in almost the same way as when the ordinary conditions exist. But, it must be remarked, inflammatory accidents almost always arouse the gouty predisposition, and give rise to an attack. We have already described the effect of traumatism in this connection. Concerning phlegmasiæ, we may distinguish three classes:

First.—The intercurrent disease (pneumonia, pleurisy, angina, erysipelas) persists for a longer or shorter time, and then gout suddenly appears; Scudamore and Day report several instances of this sort.[4] And generally this appearance of gout is regarded as a favorable sign—it is the *critical gout.* The same thing is known to occasionally occur in rheumatism. The question should then be asked, if, in such a circumstance, the outburst of an attack has not been induced by the intercurrent disease; if this has been the case, then the appearance of gout would not be a critical phenomenon.

Second.—The inflammatory disease pursues its course concurrently with gout, without experiencing any noteworthy modification. This is sometimes seen in the case of angina and pneumonia.[5]

Third.—There is an abrupt suppression of all external phenomena of gout at the moment the intercurrent malady is developed. This is a most grave symptom, and we should endeavor to recall the gout toward the extremities, though, in most instances, this results in complete failure.

III.—Special characteristics are presented by the action of certain medicaments in gouty subjects, as, *à priori*, one would be led to imagine. Thus, *lead*, when exhibited in a medicamental dose to arrest a hemorrhage, produced rapid impregnation of the system with this metal, accompanied by the bluish line along the free margin of the gums, and attacks of lead-colic.[6] And, according to Garrod and Price-Jones, *mercury* induces saliva-

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[1] "The gouty diathesis, by reason of its frequent association with renal diseases, is a very serious complication of typhus. I have never seen recovery occur in gouty patients attacked with typhus."—Murchison: A Treatise on Continued Fevers, etc., p. 227. London, 1862.

"*Pluries arthritidem anamadverti cum febre putrida fœderatam at lethali semper cum eventu.*"—Schmidtmann: Obser., Vol. iii., p. 379.

[2] Sp. Wells: Practical Observations on Gout, etc., p. 87. London, 1854.

[3] Garrod: Reynolds' System, p 855.

[4] Scudamore: On Gout, p. 21. Day: Diseases of Advanced Life, p. 317. London, 1849. Patissier: Rapport sur les Eaux de Vichy, Obs. 50, 52. 1840. Parry: Collection. Vol. i., p. 246.

[5] Scudamore: Loc. cit.

[6] Garrod: On Gout, p. 578.

tion much more speedily in gout than in other conditions;[1] and I may add that *opium* should be administered with the utmost caution to those subject to chronic gout, when they present any indication of renal disease. In these cases this drug is seen to produce cerebral symptoms out of all proportion to the dose exhibited.[2]

Let me point out another interesting fact—one belonging to the same order of ideas—namely, the failure to eliminate turpentine from the urinary passages. Hahn (quoted by Guilbert) administered this drug for seventeen months to a gouty patient without causing that characteristic odor of the urine which commonly occurs in similar instances. Was this patient suffering from albuminuria?[3]

IV.—We shall now consider those concomitant diseases of gout which present a more intimate relationship to that affection than those which we have just dwelt upon, and which are more evidently associated with the group of modifications the economy undergoes.

Hunter laid it down as a rule, that, when one diathesis invades our organism, no other general disease can exist concurrently with it; in other words, a constitutional disorder, when once it is established in an individual, admits of no rival.

From this principle arose the doctrine of *antagonisms*—a doctrine undoubtedly exaggerated by the Viennese school (Rokitansky, Engel), but which, nevertheless, is founded in truth.

Still, along with antagonisms, there certainly are affinities; and it is of these relations especially that we desire knowledge and insight.

A.—*Gout and diabetes.*—The idea of a more or less direct connection between diabetes and gout can scarcely be traced back more than two score years. Scudamore, far from suspecting this affinity, holds that these two diseases arise from distinctly opposite causes. But a German author—Stosch, of Berlin—who published (1828) "A Treatise on Diabetes," describes therein a *metastatic diabetes* which occurred after the cessation of gout, and in connection therewith quotes two English writers, Whytt and Fisher. Two years later Neumann records the occurrence of a *symptomatic diabetes* in gout.[4]

At a later period, Prout, who seems to have touched on all questions of this kind, places attacks of gout and rheumatism among the most frequent causes of diabetes. "Nothing is more common," he says, in another place, "than to find a little sugar in the urine of gouty patients, although they remain unconscious of it until the ordinary symptoms of diabetes—polyuria, thirst, emaciation—openly declare themselves."[5] Another English author, Bence-Jones, has likewise observed that *uric acid gravel* predisposes to diabetes.[6]

In France, Rayer has frequently remarked to his students the connection subsisting between uric acid gravel, gout, and diabetes. You can consult, in this connection, the thesis of M. A. Contour,[7] and the lectures of Claude Bernard. This eminent physiologist has, in fact, stated that dia-

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[1] Garrod: Loc. cit., p. 354. Price-Jones: Medical Times. Vol. i., p. 66. 1855.
[2] Todd: Clinical Lectures on Urinary Diseases, p. 343. 1857. Charcot: Gaz. Médic., Nos. 36, 38, and 39.
[3] Guilbert: De la Goutte, p. 100. 1820.
[4] Pathologie. Vol. vi., p. 607.
[5] Prout: Loc. cit., pp. 33, 34.
[6] Marchal (*de Calvi*): Loc. cit., p. 233.
[7] Contour: Thèses de Paris, p. 49. 1844.

betes may alternate with the symptoms of some other disease, and particularly with *attacks of gout* and rheumatism.[1]

We had an opportunity ourselves to observe a case which completely confirms the statements of Claude Bernard. A man, fifty-six years of age, who had been suffering from gout for a long time, had succeeded in diminishing the intensity of the attacks, and finally, in nearly making them disappear altogether, by using—rather say abusing—a specific remedy (*liqueur de Laville*);[2] but, following a slight attack which he promptly repressed, appeared thirst, polyuria, increased appetite, emaciation, weakness, and the other characteristic phenomena of diabetes. When this patient consulted us for the first time, the urine contained a considerable quantity of sugar. An appropriate diet continued for over a year produced a marked improvement in his condition, though, the diabetes having considerably diminished, a few mild attacks of gout reappeared.

Marchal (*de Calvi*) has likewise been engaged in discussing the affinity between these various diseases, as regards diabetic gangrene, since 1856. Some time later he published a work where this subject is treated with remarkable ability.[3]

According to this distinguished author, there is a *uric* acid or *gouty diabetes*, and this conclusion is in conformity with previous observations, and with absolute reality. But Marchal (*de Calvi*) may be accused perhaps of having extended the domain of this form of diabetes too far, and, indeed, that of the uric acid diathesis in general. The considerations which he presents in this respect are hardly applicable, in France at least, to any but the favored class of society.

It cannot be questioned but that an affinity exists between diabetes on the one hand, and gout and uric acid gravel on the other; although the frequency of this relationship varies according to the kind of people in whom we observe the occurrences. In this way Griesinger who has studied diabetes among all classes of society, found gout in *three* out of *two hundred and twenty-five* diabetic subjects;[4] Dr. Seegen, on the contrary, practising medicine at the springs of Carlsbad, and where the patients consequently belong to the higher walks of life, met with *three* cases of gout among *twenty-one* diabetic individuals. The proportion is thus seen to vary from one-tenth to one-seventy-fifth.[5]

Nevertheless, we must not be restricted to the registration of cases where gout is transformed into diabetes in the same individual; but the hereditary transmission of symptoms must likewise be studied, and also their distribution among different members of the same family, as in the case of diseases of the nervous system, where this method has met with so much success.

It is very rare to find gout and confirmed diabetes coexisting in the same individual; but these two diseases alternate with and succeed each other. Uric acid gravel or gout opens the scene; and then, usually, gout

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[1] Leçons de Physiologie Expérimentale, etc., p. 436. 1855: "Diabètes alternants."

[2] Dr. Laville's *anti-gout liquid* and *pills*. Active principle of liquid probably veratria—certainly a powerful remedy or poison; teaspoonful in water. Pills of salicylate of soda and physalin; two a day with meals. Shorten attacks at expense of health. —L. H. H.

[3] L'Union Médicale, No. 29. 1856. Recherches sur les Accidents Diabétiques, etc.: loc. cit., pp. 409, 469. 1864.

[4] Griesinger: Studien über Diabetes. Archiv für physiol. Heilkunde, p. 16. 1859.

[5] P. Seegen: Beiträge zur Casuistick des Meliturie. Virchow's Archiv, Vol. xxi., Vol. xxx. 1864.

vanishes the moment diabetes makes its appearance. Rayer had already observed that gout changed into diabetes, and Garrod says in these very words: "*Gout stops when diabetes appears.*" [1]

Let me add that *obesity* frequently precedes the development of gout.

In cases such as those to which we have just alluded, the prognosis is sometimes as grave as in ordinary diabetes, and phthisis pulmonalis and symptoms of gangrene suddenly occur. Still it must be acknowledged that gouty diabetes is in general relatively benignant, especially if the patient follow a suitable diet. Then it is that diabetes is latent (Prout). I might cite cases where a cure seemed to coincide with a return of gravel or gout, and this caused Prout to say that the appearance of uric acid gravel in diabetes is a favorable sign; [2] nevertheless, these two diseases may coexist and yet not mutually improve.

We shall now regard the analogy between gout and diabetes, considered in a family composed of several members; and thus we notice a father who is gouty, diabetic, and phthisical, beget a gouty son (Billard de Corbigny [3]); or, again, a diabetic father have a gouty son (personal observation).

We had an opportunity, ourselves to witness a very remarkable case of this kind, which Dr. Réal communicated to us, and where gout, scrofula, diabetes, and obesity were seen manifesting themselves in the majority of the members of the same family. This observation is here reduced to tabular form:

TABLE I.

Father, brewer, distiller...	.....	.....	A colossus..	Diabetes.....	Died pthisical (48 years old).
Mother.....	Lymphatic	Sciatica.			
First son, brewer.....	Scrofula, Keratitis	Articular rheumatism.	Obesity.....	Diabetes at 50	Still living (60 years old).
Second son, brewer.....	.....	Gout at 25	Obesity at 35	Diabetes.....	Died in delirium.
Third son	Lymphatic	Gout at 30.....	Obesity	Diabetes.....	Died of an accident.
Fourth son, alcoholic habits	.....	.....	Obesity	.....	Died of cirrhosis.
Fifth son.....	Keratitis..	Gout	Obesity at 35	Diabetes.....	Died phthisical (48 years old).
A daughter	.....	Gout	Obesity	.....	Still living.
Daughter of the latter.....	....	Gout	Obesity	.....	Still living.

There is evidently a more or less intimate connection between these different diseases which are thus reproduced, in various degrees, in all the members of the same family; and I have noticed the following combination:

TABLE II.

Gouty father.....
- First son—*gravel.*
- Second son—*diabetes.*
- Third son—*gout, phthisis.*
- Daughter—*gravel.*

Examples of this kind could easily be multiplied, but I think enough

[1] A. B. Garrod: Reynolds' System of Medicine. Vol. i., p. 825. London, 1866. See also: Gulstonian Lectures on Diabetes, in the British Journal, p. 319. 1878.

[2] Loc. cit., p. 25.

[3] Gaz. des Hôpitaux, p. 212. 1852.

has been said to show you that there is a correlation between the uric acid diathesis, diabetes, and gout, which is governed by laws as yet unknown.

It is easy to appreciate what are the practical deductions from these data. The urine of the gouty must, necessarily, be carefully examined ; and when this particular variety of diabetes shall have been recognized, a method of treatment must be established in accordance with its origin.

B.—*Gout and gravel.*—The uric acid diathesis includes gout in all its aspects, and hence it is not surprising that gravel, so often a manifestation of the diathesis, should very frequently be met with in gouty patients. The bond which unites these two diseases has been acknowledged in all times. "You have gravel and I have gout," wrote Erasmus to Thomas Morus, "we have espoused two sisters." Sydenham, Murray and Morgagni described this affinity, which seems to us beyond all dispute.

Still there is, at the same time, a certain antagonism between the two conditions ; it is rare, indeed, to meet them simultaneously, for they rather tend to succeed each other in alternation. The most frequent occurrence is for gravel to precede gout and to disappear when the latter is developed ;[1] but the converse may be observed, and in some cases gout completely disappears when gravel is established. I once witnessed a case of this kind. Besides, when gout and gravel are seen coexisting, one must not suppose, as is too often the case, that they are simultaneous phenomena, for gravel frequently accumulates for a long time in the kidney before its expulsion from the organ, thus giving rise to the symptoms of renal colic.

It is important to notice that the chemical composition of urinary concretions is not always the same in gout. Uric acid is always found, but urate of ammonia may also be present, and sometimes they are composed of oxalate of lime. And oxalic gravel is likewise closely allied to uric acid gravel, since uric acid, as you know, may be regarded as a composition of urea, allantoïn, and oxalic acid.

These variations in the nature of urinary deposits may also alternate. In those gouty patients subject to calculus, Gallois has sometimes found the concretions formed of concentric layers, in which uric acid and the oxalates were alternately deposited, thus clearly proving the changes which successively occurred in the composition of the products of renal excretion.[2]

Finally, let me remark that these two acids may be met with in the blood, sweat and urine of gouty subjects,[3] at periods other than those when gravel manifests itself—another proof of the correlation existing between these different morbid phenomena.

I think, however, that it should be mentioned here that the formation in the urine, a short time after its emission, of a sediment composed of amorphous urates or uric acid in the crystalline state, does not necessarily prove that the excretion of this acid is absolutely increased. A considerable diminution in the watery part of the urine and a markedly acid reaction are sufficient conditions for inducing precipitation of these sediments, without any real increase in the amount of uric acid. On the other hand, we know to-day, thanks to the labor of Bartels,[4] that urine which preserves perfect limpidity for a long time after its emission may contain a considerable proportion of uric acid. To know the true state of affairs, then, in

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[1] Scudamore : Loc. cit., p. 531.

[2] De l'Oxalate de Chaux dans les Sédiments de l'Urine, etc. : Mém. de la. Soc. de Biologie, p. 74. Paris, 1859.

[3] Garrod : On Gout. Loc. cit., p. 127.

[4] Harnsaure ausscheidung in Krankh. : Deutsch. Archiv für klin. Medicine. Bd. I., Heft I., p. 13. Leipzig, 1865.

such a case, it is indispensable to analyze the total amount of urine passed during the twenty-four hours, and even to repeat this examination for the five or six days succeeding, according to the teachings of Parkes and Ranke;[1] for it has been proved that the excretion of uric acid undergoes the most marked variations, not only at different periods of the same day, but also from one day to another.

It seems, however, very reasonable to admit that uric acid exists in the blood in excess, when urinary sediments form, not after, but before emission; and still more so when gravel is present. But this occurrence may be induced by causes entirely independent of the uric acid diathesis, a purely local inflammation of the urinary apparatus sufficing for its production (Brodie, Rayer).[2] More than once I have had an opportunity to discover a complete absence of uric acid in the serum of the blood of non-gouty individuals who habitually passed uric concretions of larger or smaller size during the act of micturition.

Still, I do not mean to absolutely deny a correlation between these two orders of occurrences. Far from it—for it is established that gravel, in certain patients, arises from the presence of an excess of uric acid in the blood. Dr. Ball communicated a case to me where a man, sixty-four years of age, frequently passed small uric calculi after a violent attack of renal colic. A blister having been applied to the epigastric region, it was found that the serous fluid obtained in this manner contained a considerable quantity of uric acid. This patient, however, had never had any of the symptoms of articular gout, and had no albuminuria. This case must undoubtedly be associated with those where gravel is seen to precede the occurrence of gout, and thereafter to alternate with this disease.

Indeed, we can make three classes in this connection. Sometimes gravel precedes gout; this is most frequently the case. Sometimes it follows it; this more rarely occurs. And again, rarest of all, these two conditions coincide. In five hundred cases of gout, Scudamore met with only five who had calculi: and Brodie claims never to have seen gravel in a gouty patient who had tophaceous concretions or chalk-stones.

The symptoms resulting from gravel commingle with those of gout. There may be emission of fine gravel with the urine, and a transient albuminuria; there may be renal gravel, which Rayer describes under the name of gouty nephritis; but we know there exists another form of gouty nephritis characterized by deposits of urate of soda in the parenchyma of the kidney (the English authors' *gouty kidney*). Finally, ischuria may be present in the gouty, and renal colics, gouty pyelitis, and irritability of the bladder may occur. All these phenomena which may coexist with gravel, are not necessarily the results of it, but, as we have previously seen, they all may simulate that condition.

C.—*Gout, scrofula, and phthisis.*—Does there exist, then, a real connection between gout, scrofula, and phthisis? We are little disposed to affirm this absolutely, but it is true that scrofula is frequent in those subject to nodular rheumatism. We may then question whether we ought not to assign to this latter affection what was attributed to gout. But Prout, who has carefully studied this point, admits that scrofula and gout are frequently associated, and that the children of gouty parents are predisposed to phthi-

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[1] Parkes: On Urine, p. 218. London, 1860. Ranke: Ausscheidung der Harnsaure. München, 1858.

[2] Leçons sur les Maladies des Organes Urinaires. Transl. by Patron, pp. 251, 278. Paris, 1745. Maladies des Reins. Vol. I., pp. 94, 197, 198. Paris, 1839.

sis.[1] This latter disease, quite rare in acute articular rheumatism (Wunderlich, Hamernjk [2]), is frequent in patients who have chronic articular rheumatism, while in gout, on the other hand, it rarely occurs, although diabetes, whose close relationship to gout we have just described, is, as it were, an ever open gate for the entrance of phthisis. Garrod, however, saw phthisis develop and run a rapid course in a young man who had tophaceous concretions around several joints; but this ought to be regarded as an exceptional case.[3]

D.—*Gout and cancer.*—Does gout exclude cancerous affections? Or, on the other hand, does it favor their development?

My former teacher and predecessor in the Salpêtrière, Dr. Cazalis, believed in the existence of a close connection between these two diatheses.

For myself, I can affirm as a certainty, that in nodular rheumatism cancer and cancroid growths are not exceptional occurrences. I have never had an opportunity of meeting examples of it in cases of well-authenticated gout, but Rayer describes the existence of both these diseases, if not in the same individual, at least in the same family; and a case published some years ago in an English journal [4] proves that these two conditions may be united in the same person. It was a case of cancer of the penis, with cancer-cells in the lungs and liver, occurring in a gouty subject, sixty-eight years old, who had large-sized tophi and a gouty nephritis with the characteristic infarctions of urate of soda in the kidneys. This case alone is sufficient to demonstrate that at least no absolute antagonism exists between gout and cancer.

E.—*Gout and rheumatism.*—The relations subsisting between articular rheumatism and gout have led many observers, as you already know, to proclaim the identity of these two diseases. We shall be better prepared to give an opinion upon this point when we shall have studied rheumatism; and hence we reserve this discussion for another lecture.

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[1] Prout: Loc. cit., p. 492.
[2] Wunderlich: Patholog. und Therapie. Bd. iv., p. 578.
[3] Garrod: On Gout, p. 578.
[4] Budd: Lancet, p. 482. 1851.

LECTURE IX.

ETIOLOGY OF GOUT.

Summary.—Study of the Conditions which Govern the Development of Gout—Suitable Method to follow in Making this Kind of Investigation—Inconvenience of the Premature Intervention of Chemical and Physiological Theories—Necessity of Separating Acquired Facts from the Hypotheses which have been advanced as Applicable to them—Historical Pathology of Gout—Antiquity of this Disease—Writers who have Described its Existence—Diminution of Gout in Modern Times—Permanence of its Characteristics—Modifications arising from our Hygienic Habits and their Probable Consequences—Medical Geography of Gout—Its Residence especially in England and in London—Met with, however, to a less Extent in some other Countries—Almost wholly Disappears in Hot Climes—Analytical Study of the Causes of Gout—Causes in the Individual: Spontaneity—Heredity—Sex—Age—Temperament, Constitution—Hygienic Causes: Climate—Overeating, Want of Exercise—Intellectual Work—Venereal Excesses—Fermented Liquors: Ale, Porter, Wine, and Cider—Exciting Causes.

Appendix.—English Beers.

Gentlemen:—Hitherto we have studied gout in relation to the lesions which accompany the symptoms which characterize, and the affinities which bind it to other diseases. There now remains for us to seek the conditions presiding over its development; we shall therefore, in a few words, point out the method we purpose to follow in the course of our investigations.

We shall first begin with the empirical study of facts furnished us by direct observation, outside of all theoretical prejudices. Then we shall endeavor to interpret these data from the standpoint of modern physiology; in other words, we shall try to follow, in their successive development, the modifications which the organism may undergo through the influence of those causes from which experience teaches us gout arises. Afterward it will be necessary to ask ourselves how the changes thus occurring in the economy can induce the various phenomena that make up the clinical history of this disease; in a word, we shall endeavor to obtain an idea of the *pathological physiology* of gout. And this is the crowning work in all nosological study.

It must be acknowledged, however, that the rigorous and systematic separation of the two standpoints we have just described is far more requisite in the question we are discussing; for it is here, especially, that the premature and rash intervention of chemical and physiological theories to account for the morbid phenomena, may tend to bring this kind of study into unmerited discredit.

He who shall succeed in harmonizing the pathology of the ancients and the physiology of the moderns, will surely, says Boerhaave, deserve the highest praise.[1] But *modern* physiology in Boerhaave's time scarcely cor-

[1] Nec in medicum plus laudis redundare posset, quam ex eo labore, quo veterum pathologiam redigeret ad neotericorum physiologiam.—Boerhaave: Med. Stud. Medic. Pars, ix., Pathologia, p. 573.

responds to the science as we understand it to-day; and so, in the coming centuries, may contemporaneous physiology likewise become as obsolete in its turn. Hence, we must use the utmost reserve, and advance along this path only after infinite precautions; for what our predecessors lacked was not an appreciation of the important rôle which physiology plays in medical studies, but broader and more exact notions concerning those difficult problems they sometimes attempted to solve without having first measured their extent.

Indeed, it must be acknowledged that the history of the products of disassimilation still remains obscure, notwithstanding the progress we have made in the study of the nutritive functions; and uric acid, especially, is no exception to this rule. We know very little concerning the conditions which preside over its regular formation, and the pathological circumstances that may modify it. Thus, at the very commencement it is easy to foresee that the pathogenesis of the uric acid diathesis is still in a rudimentary condition, and that, consequently, at the present time it is impossible for a complete theory of gout to be drawn up; at the utmost we are only permitted to place a landmark here and there, which may, perhaps, serve to direct the investigations of those who come after us.

We shall begin this analysis with a rapid glance over the *history* and the *geography* of gout, for, since we are concerned here with a constitutional disease, and one essentially arising from the general state of the individual, it is absolutely necessary to study in order that we may obtain a firm grasp on its characteristics, the climatic and social conditions which, collectively, seem to predispose the human species to this malady. To regard a disease in this light is to construct its etiology on a grand scale.

I.—Historical Pathology of Gout.

To recount the vicissitudes which diseases have experienced in the course of centuries, and to search in history for the causes of these changes—this is the chief aim of historical pathology; and besides, investigations of this kind enable us not only to appreciate the pathogenic influence of external causes, but also the results of the inherent conditions of man himself.

But, in order that it may be possible to apply this process to the study of a disease, the attention of our predecessors must have long since been called thereto; and it is only in these cases that one may hope to gather an ample harvest from historical documents. Now, similar conditions are rarely realized, except in the case of epidemic diseases, which used to produce such terrible ravages—and of certain chronic affections which, in all times, have attracted the notice of investigators. A few examples will enable you to readily grasp my idea. The plague, so formidable in past times, appeared, for the last time in France, at the beginning of the previous century (1721); it is tending, also, to become extinct in those very countries which have always been its principal centres. One may, then, with reason demand what the conditions were which, in past times, favored its development, and which to-day seem to have ceased to exist. Leprosy was still extant in Martigues at the end of the last century, but since that epoch it has disappeared from French soil; it is becoming rarer and rarer in Europe, tending to take refuge in Norway, as the gout does in England. We can readily understand how much attraction for the philosophic physician the history of an almost extinct disease may have, after it

has played so great a part and occupied such a prominent position in the minds of legislators of every century.[1]

Among the diseases whose historical study offers a truly scientific interest, gout clearly stands in the first rank; indeed, it is certain that this disease used to prevail in a somewhat epidemic manner among the more favored classes of society. To-day it is seen gradually to become extinct, although it has not undergone any change in its symptomatic development since the most remote epochs, inasmuch as we find it completely described in the works of the ancients.

We shall now give you a succinct statement of the principal data furnished us by history for the solving of this question.

Antiquity of Gout.

There is no doubt but that gout has been known in Europe since the days of remotest antiquity; the writings of Hippocrates are proof of this. But it is in the reign of the first Cæsars that it seems to have reached its culmination; and in this connection we possess unexampled accounts that leave nothing to be desired. The writings of physicians, the works of historians, the satires of poets—all are filled with allusions to this malady.

In the first century of the Christian era, Aretæus and Celsus on the one hand, and Ovid and Seneca on the other, have given ample information concerning the pathological condition of the Roman people in this regard. In the second century, Galen (130 A.D.) and the interesting dialogues of Lucian of Samosata[2] furnish us with details valuable both from a hygienic and a medical standpoint. In the third century an edict of Diocletian exempts gouty subjects from public services when they are attacked with articular deformities great enough to interfere with the exercise of the ordinary functions of life, a circumstance which seems to prove both the extreme frequency of gout at that period, and the immutability of the chief symptomatic characteristics of this disease.

From the third to the sixth century things seem to have remained unaltered, if we may judge from the writings of Oribasius (326–403 A.D.), Alexander of Tralles (525–605 A.D), Aëtius, Paulus of Ægina (660 A.D.) and many other physicians. In the middle ages the Arabians, continuing the medical traditions of antiquity, inform us that gout has lost scarcely any ground since that epoch[3]; and the authors of the Lower Empire (*Byzantine*), Actuarius, Demetrius Pepagomnenus, etc., carry us up to the thirteenth century. Modern times being reached, we find ourselves confronted by

[1] The stringent measures which inflicted upon the leper an absolute isolation from all society were maintained with unrelaxed severity during the middle ages. They may have contributed to the extinction of leprosy. Certain pathologists prefer to invoke, in these cases, a morbid spontaneity, and we willingly accept the term, provided it be thoroughly understood that it prejudges in nowise the real gist of the question, and only serves to express a gap in our knowledge.

[2] Tragodopodogra; Okupous the Fleet-footed. The latter poem, according to some critics, should not be attributed to Lucian. *Okupous*, one of Homer's names for Achilles.—L. H. H.

[3] It must not be forgotten, however, that the information offered by the authors of that period is not always worthy of absolute trust. Too often they copy from one another, without thinking of collecting personal observations. The Arabians, especially, have borrowed largely from Greek medical literature; and as gout is frequently discovered in the works of the ancients, they certainly appropriated a considerable part of their labors.

innumerable evidences that leave no doubt as to the general diffusion of gout over Europe.

And thus, gentlemen, you see an uninterrupted chain of historical proofs testifying that for more than twenty centuries this disease has held sway in the countries we now inhabit. But to-day we need only glance around us in order to convince ourselves that gout is becoming rarer and rarer; and here it is proper to enter into a few details.

Diminution of Gout in Modern Times.

This retrograde movement of gout seems especially to have manifested itself since the beginning of the present century. Documents collected by Corradi[1] inform us that even in England this disease is diminishing in frequency, according to Owen and Fuller; that it has markedly declined in Holland, according to Coley; and also in Switzerland, according to Professor Lebert. It has almost disappeared from those localities where it used to prevail, for in our time it is scarcely ever met with in Rome or Constantinople. In this respect it is evident that the state of affairs has undergone considerable change. And this, undoubtedly, is the reason why the works which have appeared upon this subject within the last sixty years are so few in number; for, save in England, materials for new investigations in gout are very rarely obtained from observation.

In spite of its decadence, however, this disease has undergone no change in its symptomatic developments, as you will soon see.

Permanence of the Characteristics of Gout.

We have only to compare the descriptions bequeathed to us by antiquity with those found in modern works, to convince ourselves that, from a clinical standpoint, gout has always remained true to its primitive type. Okupous the fleet-footed[2] completely resembles, in this respect, the cases observed by Van Swieten, sixteen hundred years later.

Concerning the etiology, we are always confronted by the same conditions. Suetonius called gout *morbus dominorum*, and Sydenham has expressed the same idea in slightly different terms. As for the influence exercised upon the development of this disease by excesses at table, this has always figured in the universally accepted class of traditions.

And finally, let us add that the Greek and Roman physicians, who described the characteristics of gout so concisely, barely mentioned the existence of articular rheumatism; so that many authors consider it a new disease, or at least as one almost unknown in ancient times. This is the opinion of Sydenham, reiterated later on by Hecker and Leupoldt.[3]

Farther on we shall have an opportunity to prove to you, by unexceptionable evidence, that there is much exaggeration in this way of looking at it, and that rheumatism most certainly existed among the great nations of antiquity; but in this case there was surely quite a strange contrast, and one which suffices, at all events, to show that the general physiognomy of gout has never varied.

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[1] Della odierna diminuzione della podogra, etc., del Dre. Alfonso Corradi. Bologna, 1860. [2] Achilles.

[3] Hecker: Rede über die aufeinander Folge der Dyskrasien, etc., in der Med. Ver einzeit, 1837. Leupoldt: Geschichte der Medicin., p. 66. Berlin, 1863.

What deductions may we draw from the facts we have just set forth? Shall we admit, with Corradi, that the decadence of gout results from moderation in our customs and from a better alimentary hygiene? There certainly has been a great change in our habits in this respect. The suppers of Lucullus have disappeared for many a century; we no longer possess the heroic appetites of the doughty knights of the middle ages; nor is it the fashion to-day to gather, as at the feasts of the Burgraves,

"Autour d'un bœuf entier, servi sur un plat d'or." [1]

We are accustomed to a less abundant diet, to one consisting less exclusively of animal food, and our repasts are not so prolonged; besides, the abuse of fermented drinks has considerably diminished, even in England, where the customs of the last century left much to be desired in this respect.

II.—Medical Geography of Gout.

Medical geography is, equally with historical pathology, one of the most fruitful means of investigation in etiological research. It enables us to become acquainted with the different regions of the globe in which certain diseases prevail, and thus allows upon the grandest scale, a study of the cosmical, tellurial, and even anthropological conditions that may favor or hinder their development.

Concerning gout in particular, geography teaches us that to-day it only exists upon one spot of our globe in the condition of a generally diffused malady: this, of course, is in England. Here, however, England proper is meant, for neither Ireland nor Scotland is in the same position, in this regard, as the southern portion of the United Kingdom. Besides, it is in London especially that the predominance of gout is shown: in that city it prevails, not only among the best classes of society, but even among the body of the people and working-classes least favored in respect to the necessaries of life. We shall endeavor, farther on, to explain the reason of this singular choice of habitation. We will merely say, for the time being, that gout exists in other parts of the globe, though in a very much less degree. It is met with in some portions of France, especially Lorraine and Normandy, the provinces which have in all times been noted for their good fare. It is likewise present in Germany, and in countries where beer is the ordinary beverage of the population.

It is certain, moreover, that this disease is only prevalent in the temperate regions of the earth. Near the equator and in the tropics, gout is hardly known; in India it sometimes attacks Englishmen, though less frequently than in their native country, while it spares the indigenous population. In Egypt it only attacks the Europeans and those comfortably conditioned Turks who set at naught the precepts of the Koran; but the fellahs seem to enjoy absolute immunity.

Finally, in Brazil they hardly know what gout is, although the diet of the inhabitants is very highly animal (Dundas). I have taken most of these details from Dr. Hirsch, of Berlin, who has published an excellent work upon this subject.[2]

Climate has a most obvious influence here: it is not a question of race, for the negroes in the English army, when placed under the same conditions

[1] Around a whole ox, served up on a golden dish.
[2] Handbuch der historisch-geogr. Pathologie. Erlangen, 1859.

as the whites, are, like the latter, liable to contract gout; this seems to be proved by some observations reported by Quarrier.[1]

Articular rheumatism behaves in a very different fashion in the above respect: it appears to exist in all climates, being often met with in India in the acute, as well as the chronic form: indeed, to use an expression of Mühry's, rheumatism is an ubiquitous disease.[2] Here is a striking difference between these two analogous diseases; and one which it is important to point out.

We have only sketched the most salient points of the pathological history and medical geography of gout. Now, however, we must abandon this "straightforward" method of study, descending from the very general standpoint we have taken in order to engage in the minute analysis of those particular circumstances which may give rise to this disease. In the course of this study we shall have an opportunity to point out facts that are still but little known in France, and which, in every respect, merit your attention.

ANALYTICAL STUDY OF THE CAUSES OF GOUT.

I.—Causes whose Seat is in the Individual.

A.—*Spontaneity.*—It is a point beyond dispute that gout can develop spontaneously; facts adduced by all authors prove it, and I have myself met with such cases. There reside, then, in the very constitution of some individuals, conditions which are favorable to the development of gout, and external circumstances do nothing but bring out the disease. In this there is nothing that should astonish you: for an excessive production, or a faulty elimination of uric acid seems to be the fundamental condition for the gouty diathesis. We find uric acid a normal ingredient in the circulatory fluid, and for ever so slight an increase in this quantity, the whole train of pathological changes may be displayed.

B.—*Heredity.*—The definition of gout as expressed by modern writers always entails the idea of heredity. Physicians who collect observations from hospitals have already established the truth concerning this point, namely, the frequency of hereditary transmission; and in private practice this influence of heredity is recognized *à fortiori*. I shall submit a few statistics to you, which may impart an approximate idea of the importance of this condition.

Scudamore met with heredity in 309 out of 523 cases, or in a little over fifty-nine per cent.

Patissier (*reports*) 34 times in 80 cases, or in more than forty-two per cent.

And Garrod in 50 out of 100 cases, or in one-half.

Hereditary gout often develops quite early, and before the time this usually happens from other causes. Now, ordinarily, gout of a spontaneous origin makes itself manifest between the thirtieth and the thirty-fifth years of life; but hereditary gout does not wait so long a time before showing itself. It often makes its appearance at one definite age in each member of the same family. Garrod records a case where, in one of the great

[1] Edinburgh Med. and Surg. Journ. Vol. ii., p. 459. 1808.
[2] Klimatologische Untersuchungen, p. 212. Leipsig, 1858.

English families, the eldest son was attacked with gout the day he received the ancestral heritage; and, moreover, this succession was perpetuated during four centuries.

C.—*Sex.*—The influence of sex upon the production of gout is not less evident than that of heredity. In this respect women enjoy a comparative immunity impossible to deny. Of the eighty cases collected by Patissier, there were only two belonging to the female sex. It is at the time of the menopause that these phenomena occur—a fact observed by Hippocrates.

Now, as we shall soon see, chronic rheumatism essentially differs from gout in this regard. There are, however, exceptions to the rule, and women are sometimes seen to become subjects of gout quite early in life; but here we will almost always recognize the influence of heredity.

Finally, let us add that it is the asthenic form of the disease, especially, that prevails in the female sex.

D.—*Age.*—From the thirtieth to the thirty-fifth year of life is, according to Scudamore, the classical age for gout. It is very rarely seen before the twentieth, or after the sixtieth year. Garrod, however, met with it once in a nine-year old patient, and once in a young man under seventeen years of age. He also reports a few cases where this disease was developed in men from sixty to seventy years old.

Rheumatism, on the other hand, appears earlier, and is generally observed before the age of thirty-five.

E.—*Temperament, constitution.*—It has frequently been attempted to collect together characteristics of a special constitution predisposing to gout; but the study of facts tells us that it respects no temperament, and may develop in the feeble as well as in the most vigorous constitutions. The type of the disease, however, is modified by the general state of the organism; the sthenic form occurs especially in the sanguine and plethoric, while the asthenic variety is met with in women and those of a nervous temperament.

II.—Let us forego, now, the further study of the causes residing in the individual, to consider those resulting from his surrounding circumstances, first discussing hygiene, and especially alimentation, since in this way we shall obtain valuable data for the solution of the problem we are endeavoring to elucidate.

A.—*Climate.*—Medical geography has already demonstrated that gout belongs solely to the temperate regions of the earth, to all appearances avoiding the tropics. Unknown in Brazil, Africa, and the equatorial regions, it sometimes, however, attacks those Europeans who continue the habits of a cold climate while residing in a hot country; and this is why the English in East India are often liable to be attacked with it.

B.—*Excess in eating and want of exercise*—It has always been recognized that too nourishing a diet and too idle a life—two causes frequently acting in concert—directly predispose to gouty manifestations; and this is undoubtedly the reason why it prevails among the higher classes, and is less frequently met with among the masses. This is familiarly expressed by saying that gout proceeds from an excess of receipts over expenses. We shall soon see that facts do not sustain such a simple interpretation as the above; it is, at least, certain that a diet consisting of too much animal food favors the development of this malady, and that large eaters are frequently among the number of its subjects.

C.—*Influence of the nervous system.*—We may no longer deny the influence exercised by cerebral causes; and intellectual labor, moral emotion,

and intensity of thought have always occupied an important position in the etiology of gout. This justified the witty *mot* with which Sydenham consoled himself for being a sufferer from gout: "*Divites interemit plures quam pauperes, plures sapientes quam fatuos,*" are the words he uses in speaking of this disease.[1] It is indisputable that the most distinguished political characters in England, at least, were martyrs to this affection; we may cite, among others, the case of the two Pitts. The first of these two great ministers—the Earl of Chatham—was known to be no worshipper of Bacchus; while the same cannot be as truly said of his son, William Pitt, who never spoke in the House of Commons without having first fired his eloquence with copious libations.

D.—*Venereal excesses.*—Abuse of venery may clearly act in an unfavorable manner upon gout, on account of the perturbation of the nervous system which results from it. But we have recourse here to a much simpler explanation: it is well known that excesses of this sort are closely connected with the carouses succeeding feasts, and it is to the co-operation of the latter occurrence, perhaps, that we must here assign the principal rôle.

Two other causes remain to demand our attention; I refer to the influence of fermented drinks and of saturnine poisoning.

The action of fermented liquors is so evident, that Garrod has well said: "Man, deprived of these beverages, would never have known the gout."

The province of lead-poisoning is, in this respect, much more limited; but, from a standpoint of pathogenesis, this aspect of the question is of the deepest interest.

We shall consider, in order, these two classes of phenomena.

E.—*Fermented liquors.*—From our point of view, we must make a radical distinction between spirituous liquors (rum, brandy, whiskey, gin, etc.)—liquors which contain from forty to seventy parts of alcohol in one hundred—and the simple fermented beverages (wine, beer, cider, etc.), whose alcoholic strength varies from four to twenty per cent. At the first glance it looks as though the more a liquor is charged with alcohol, the more it predisposes to gout; but such is not the fact, and you will be astonished to learn that the use—even the abuse—of distilled liquor does not seem to exercise the slightest influence in this respect. Indeed, gout is hardly ever met with among people who drink brandy. In Sweden, where alcoholismus is so frequent—according to Magnus Huss—this disease is out of the question; and it is the same in Denmark, Russia, and Poland. In Scotland and Ireland, gout is rare among the lower classes. In Edinburgh, with a large hospital practice, Bennett[2] and Christison met with barely more than one or two cases of it. Now, in these countries, the only alcoholic liquor which the people drink is whiskey.

In London, on the other hand, gout is a very common disease among the working classes, and is frequently met with in the hospitals. Now, the only fundamental difference that can possibly be established, in this respect, between the northern and the central portions of the United Kingdom, is the enormous consumption of strong beer (ale, stout, and porter) by the laborers who live in the metropolis.[3]

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[1] It kills the rich oftener than the poor, the wise man oftener than the fool.

[2] Clinical Lectures, etc., by J. Hughes Bennett. Second edition, p. 916. Edinburgh, 1858.—L. H. H.

[3] See Appendix, inserted at the close of this lecture.

This truly remarkable influence of these beverages has been recognized by all English writers on the subject, commencing with Scudamore. He tells us that "gout is much more frequent in London, among the masses, since the use of porter has become habitual." The testimony of Watson, Budd, and Todd also corroborates this assertion: "Most of those who are given to the use of beer, especially porter, sooner or later suffer from gout," says the last-named of these three authors.

An example borrowed from Budd[1] will illustrate the influence which this kind of liquor exercises. There is, in London, a body of laborers who work at the raising of ballast from the bottom of the Thames. This is done during low tide, and consequently, the working hours fall sometimes during the day-time, and at other times at night. The workmen, who are exposed to all sorts of inclemencies, are also obliged to expend great muscular effort; and, in order to obtain the best return (please to notice here the practical nature of the English), a large allowance of porter is given to these men. Each one drinks two or three gallons a day; and, apart from this enormous consumption of fluid, their diet is that of the very lowest classes in London.

Now, gout is an exceptionally frequent disease among these poor men, who share this sorry privilege with the peers of the realm; and although their numbers are but very few, many of them are each year admitted as gouty subjects in the Seamen's Hospital. And yet, these are generally unfortunate Irish peasants, in whom an hereditary vice of constitution could not be advanced as a cause.

Garrod, on his side, attained the same results. He states that the *employés* of large breweries are frequently attacked with the gout, and yet nothing can be found in their antecedents to explain this morbid predisposition, except it be the abuse of ale, and porter especially.

These two beverages, however, are not remarkable for their richness in alcohol; according to Mulder, Scotch ale contains eight per cent. of alcohol, and porter five per cent.[2] This proportion is smaller than that in our French wines, and does not exceed that of the German beers, which seldom produce such effects, in spite of the large amounts drunk in the breweries.

It is evident, consequently, that *à priori* reasoning cannot be applied to the question we are discussing, and that the influence of fermented drinks upon gout is far from corresponding to their percentage of alcohol. Circumstances of a different kind, which have escaped us to this day, probably interpose themselves at this point; and for every kind of liquor we must have recourse to the data of direct experimentation.

We shall now consider the action of wine. The first rank must here be conferred upon the spirituous wines (Port, Sherry, Madeira, Marsala), which are so extensively used in England, in all classes of society. These contain a considerable quantity of alcohol, varying from seventeen to twenty per cent.

The lighter wines (Rhine, Moselle, Bordeaux, Champagne) are far from exercising the same influence as the former class.

But we cannot say the same of Burgundy, which nevertheless, contains scarcely more alcohol than the preceding.

"*Red Hermitage* and *Burgundy*, the latter especially," Scudamore says, "contain gout in every glass."

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[1] Tweedie: Library of Medicine. Vol. v., Art. Gout. Also in Bennett's (J. Hughes) Clinical Lectures, etc., p. 992. Edinburgh, 1868.—L. H. H.

[2] Mulder: De la Bière. Trad. Delondre, p. 327. Paris, 1861.

Even cider, that beverage seemingly so free from danger, also appears to favor the development of gout. According to Garrod, it is soft (*new*) cider, and that having undergone only partial fermentation, which possesses this unpleasant property.

I think that, as the influence which certain beverages exercise in this regard has been sufficiently well proved, we may now safely pass to the consideration of another subject.

B.—*Lead-poisoning.*—Garrod states that out of fifty-one gouty patients who were in his service at the hospital, no less than sixteen were painters or plumbers by trade ; and subsequent researches only confirm this strange result. Thus, saturnine impregnation has been ranked among the predisposing causes of gout.

This coincidence once pointed out, documents in its support flowed in from all quarters. Among the authors anterior to Garrod, Musgrave may be cited as one who noticed gout following lead-colic, Falconer as another making the same observation, and Parry, who, in his collection of cases, has shown that gout is frequent in those attacked with lead-paralysis. Finally, Todd reports several cases of gout occurring under analogous circumstances.[1]

Since the publication of Garrod's work, several English authors have described cases of this kind ; we may especially cite Burrows and Begbie.[2] But in England we must take into account those alimental causes we have just enumerated. In France, where lead-colic is so common, how does it happen that gout is so rare among the great mass of the population ?

Well, in cases of saturnismus there are a few patients with the gout in whom lead-poisoning is the only cause that can possibly be adduced. We have ourselves had an opportunity to observe a very remarkable case of this kind, and Dr. Bucquoy has just reported an almost identical case in Charity Hospital.

It now remains to determine the cause of this singular coincidence. Garrod affirms that an impregnation of the system with lead leads to an accumulation of uric acid in the blood, especially in advanced cases where there is paralysis ; this fact has been established in non-gouty cases of saturnismus, which seem not to have been albuminurious ; for their urine had been examined, and it had been determined that the proportion of uric acid was sensibly diminished ; but in these analyses there was no question of the presence of albumen. Garrod asks himself whether, in this case, there was an over-production of uric acid, or a failure in excretion of this product. He leans toward the latter hypothesis, and this is the experiment on which he bases his view : after having for several days examined the urine of a certain number of patients suffering from various diseases, in order to establish the normal proportion of uric acid, he commenced to employ acetate of lead medicinally with them, and he found that the excretion of uric acid diminished.

It is then by paralyzing the action of the kidney, at least so far as concerns the elimination of uric acid, that lead favors gouty manifestations ; but can the disease arise from the influence of this cause alone ? In a few

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[1] G. Musgrave : De arthritide symptomaticâ, c. x., art. 5, p. 65. Genovæ, 1725. C. H. Parry : Vol. i., p. 243. London, 1825. Todd : Practical Remarks on Gout, p. 44. London, 1843.

[2] W. Falconer : Brit. Med. Jour., p. 464. 1861. Begbie : Edinburgh Med. Jour., p. 128. August, 1862. Charcot : Gazette Hebdom., p. 433. 1863.

exceptional cases perhaps it can; but if there are any adjuvant causes, the effects of lead will be exhibited last of all.

III.—*Exciting causes.*—Incapable themselves of producing gout, still the conditions we are about to enumerate possess great potency in bringing about the development of an attack.

A.—*Alcoholic beverages.*—The ingestion of but a small quantity of certain wines (champagne and port, for example), in the case of gouty patients, is enough to induce at one time a violent attack of gout, and at another simply a swelling of the great toe. Hence, Garrod has well said: "In whatever individual a few glasses of wine are always sufficient to rapidly and invariably provoke an inflammation in a joint, that inflammation is certainly of a gouty character."

B.—Attacks of *indigestion*, and gastric derangements act in a similar manner.

C.—*Wet cold* and suppression of the perspiration are in the same category.

D.—Excessive *intellectual labor*, to which we have already alluded as a determining cause of gout, is included also in this enumeration.

E.—*Traumatic* causes, operations, fractures, etc., act in like manner, and I have seen a wound simultaneously induce an attack of trismus and one of gout.

F.—*Debilitating* causes—hemorrhage, bloodletting, and grave diseases—also exercise their influence upon the production of an attack. It is all the more interesting to notice this point, since people are fond of painting gout as a disease of plethoric individuals; but Todd has shown that it readily attacks debilitated subjects.[1]

We shall devote our next lecture to the theory of gout.

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[1] Recently I had under my charge a former officer in the Confederate Army, who, during the rebellion in the United States, was made prisoner by the Union troops. Confined in a damp and unhealthy prison, and having a very insufficient diet, he became a subject of gout; he remains so till this day, and yet he has no hereditary antecedents that could predispose him to gout, and previous to that time he never had the least indication of the disease.

APPENDIX TO LECTURE IX.

English Beers.

The question of the influence exercised by *ale* and *porter* upon the development of gout is one that frequently recurs in these lectures, and it seems to me to be necessary to present, in this connection, some description of the processes employed in making these beverages, and the chief properties that characterize them; so I requested Dr. Ball to prepare for me a short account bearing on these points, which I here present to the reader. The information therein is all the more useful, since it can be found in no medical work published up to this time.

It is beyond dispute that, from the remotest antiquity, people who were not acquainted with the use of wine discovered the means of utilizing germinated barley (*malt*) in order to procure alcoholic beverages.[1] Long before they left their forests the Germanic tribes possessed this art, and thus we are not astonished to find beer naturalized in England from the time of the Anglo-Saxon conquest. The laws of Ina, King of Wessex (a country of the Western Saxons), which were promulgated in 728, already make mention of *ale* and *alehouses;* and from that epoch beer has never ceased to be the natural beverage of the English.

But during the course of these many centuries public taste has varied more than once, and the brewers have been obliged to follow the fashion, except, indeed, when they have forestalled it. In the middle ages hops were not employed in beer-making, the beverage seeming to have had an insipid and sweetish taste; and they endeavored to correct this fault by the addition of infusions of bitter and aromatic herbs. It was in 1524 that the Flemings caused hops to be introduced into English brewing; but this practice was not legally authorized until 1552. The name *ale* was then given to the sweeter beverages prepared from malt (*germinated grain*), while the term *beer* was reserved for those impregnated with the bitterness of hops. In the seventeenth century, however, every vestige of this distinction was swept away, and hops were universally employed in the English breweries.

The origin of *porter* is of much more recent date. It was in 1730, according to Malone, that it commenced to be used for the first time. About

[1] Herodotus and Diodorus (of Sicily) tell us that the Egyptians knew how to make beer. Pliny and Tacitus testify the same as regards the Germans. "Potui humor ex hordeo aut frumento in quamdam similitudinem vini corruptus."—Tac.: De situ, moribus, ac populis Germ., cap. xxiii.

that time the workingmen of London were in the habit of drinking, in the alehouses, a mixture of beer, ale, and small beer, which they called *three threads*, because for every pint he drew for a customer, the inn-keeper was obliged to go to three different casks. In order to avoid this inconvenience, the brewer Harwood thought he would make a beverage that would combine the flavor of these three; he succeeded admirably, and the success attained by the new drink among the lower classes of the metropolis was such that the name *porter*[1] was given it, a name which it has retained till this day.

To please the popular taste, they formerly gave this porter a very deep color, by prolonged torrefaction of the grain. But it was soon perceived that, by doing this, they destroyed the greater portion of the saccharine matter contained in the malt, and that the richness of the liquor in regard to fermentable principles was diminished thereby. Recourse was then had to a number of artificial processes for coloring porter, which were prohibited in 1816 by an act of parliament; and the only ingredients which, to-day, serve for the manufacture of beer, are water, malt, and hops.

It was then discovered, however, that complete torrefaction of malt, though destroying the sugar it contained, gave rise to a very soluble coloring matter: and since then this substance, which came within the terms of the law of 1816, was largely employed in making porter.

To-day this beverage is a mixture of several kinds of beer, kept a long time after being commingled, so as to push fermentation to its utmost limits and convert all the sugar into alcohol. But, since the malt is exceedingly torrefied at the commencement, it contains but little glucose at the very moment when the working begins, and consequently can never be as rich in alcohol as the other varieties of beer. Its essential characteristic, however, is a tendency to acetic fermentation, for, all the sugar being destroyed, one more step suffices to convert the alcohol into vinegar. From a theoretical standpoint, this change ought never to take place; but practically the porter delivered for use is frequently acid, a fact of which I have many a time assured myself.

The name *entire* is generally given to beers that have been mingled; while *stout* is applied to one that is prepared with more care and is destined for more delicate consumers, though it participates in the general characteristics that we have just described.

Under the name *ale* are classed all the other varieties of beer that do not possess a deep color, and are not prepared from excessively roasted malt; hence, they are richer in saccharine matter and alcohol; and as fermentation has not progressed far enough to destroy all the sugar they contain, they possess a very different flavor from that of porter, and present no tendency to sour.

Thus, we may divide into two great classes the beer that is used in the United Kingdom: the *first* class is rich in color, but poor in alcohol, deprived of sugar, and ready to undergo acetic fermentation; they are also impregnated with a principle obtained by the torrefaction of grain, and which perhaps is not alien to their pathogenic properties. To this class belong the beverages known under the generic name of *porter*, and whose use so markedly predisposes to gout.

The *second* class, on the contrary, though poor in color, are rich in alcohol, and do not contain a trace of acetic acid.

It is understood, of course, that in this brief account we could not include

[1] "Probably because the London *porters* first used it."—Webster.

all the variation to which caprice, chance, or local customs may have given rise. In England, the beer of one county or shire does not resemble that of its neighbor; and each distinguished brewer has his secrets, which especially stamp the products of his manufacture. It is enough, then, to have presented the reader with a general view of the subject, without entering into a minute study of details.

LECTURE X.

PATHOLOGY OF GOUT.

Summary.—Rational Theory of Gout—It can hardly be Formulated in the Present State of Scientific Knowledge—Cullen—Discovery of *Lithic Acid* (Uric Acid)—Influence of this Fact upon Modern Works—Garrod's Researches—He establishes the Fact that Uric Acid exists in Excess in the Blood of Gouty Patients—Origin of this Excrementitious Product—It is still but little known—Are Urea and Uric Acid Immediate Products of Disassimilation?—Experiments of Zalesky.

Empirical Researches—Effects of Fasting—Animal Diet—Exercise—Contradictory Results in this Respect—Influence of Liquors: Experiments of Böcker.

Theory of a Gouty Attack (Fit)—The Articulations preferably affected—Fibrous Tissue, Cartilage—Predilection of Gout for the Great Toe—Successive Invasion of Joints—Tophi—Deposits of Urate of Soda in the Cartilages—Pain—General Reaction—Visceral Phenomena—Insufficiency of our Knowledge in this Respect at the Present Day.

GENTLEMEN:—After having passed in review the various causes which are closely or remotely associated with the production of gout, there remains for us to seek a rational theory of this disease, and to combine the data of physiology with those furnished us by clinical observation. We may not, besides, flatter ourselves that we can obtain complete success in this direction; for, even though we know the morbid principle whence arises, in this case, the pathological series of events, we are yet far from grasping all the links in the chain; the conditions presiding over the formation and elimination of uric acid are still unknown to us, and will, no doubt, long elude our investigations.

In order to understand, however, the present state of the question, it is necessary to trace the various phases it has passed through during the course of years until the present day. Let us see, then, what the opinion of our predecessors was in this respect.

The theories which were formulated concerning gout during all the seventeenth and part of the eighteenth century were essentially associated with humorism; this is always the theory of Sydenham, in slightly different words. There is morbific material in the body, the result of imperfect digestion occurring either in the *primæ viæ* or in the secondary apparatus, and the efforts of nature to eliminate this *peccant material* (phlegm, bile, tartar) constitute the symptoms of gout.

But a reaction against these old ideas set in from Cullen's time. This celebrated author held that there was no proof of the existence of a morbific material in the blood. He regards tophi, adduced by the humorists in support of their theory, as purely accidental occurrences. For him, gout results from a sort of plethora, with loss of tone in the extremities.

The progress of chemistry stepped in to modify, to a certain extent, this method of regarding the disease. In 1775, Scheele discovered *lithic acid* (uric acid) in urinary calculi and in the urine; in 1793, Murray Forbes,

by reason of the analogies subsisting between gout and gravel, promulgated the view that uric acid existed in the blood of the gouty; in 1797, Tennant and Wollaston established the composition of tophi to be of urate of soda.

Cullen's theory, however, still held sway in England. Scudamore continued to regard gout as a sort of plethora without any relation to the excess of uric acid in the blood; he considers tophi as exceptional phenomena in gout, having found them in but forty-five out of five hundred gouty subjects. Barlow and Gairdner share this opinion; and recently Barclay[1] has again recurred to this point, relying, it must be said, much more on sentiment than upon observation. But Parkinson, Home, and Holland are identified with the uric acid theory.

In France, gout has been studied only by a limited number of writers; but those who have devoted themselves to this subject admit, at least theoretically, the presence of uric acid in the blood, and fully understand the importance of this cardinal fact. In this connection we may especially cite Andral, Rayer, and Cruveilhier;[2] the latter regards the deposit of tophaceous matter in the interior of the articulations and in their vicinity as the characteristic lesion of gout. Now, these tophi consist of urate of soda; and Cruveilhier is thus led, he says, in spite of himself, to the theory of Sydenham and the older investigators: he regards urate of soda as the material cause of gout, and he considers that there is no doubt but that the first attack of gout coincides with a secretion of this product, which is repeated afresh with each subsequent attack of the malady.

Notwithstanding all the interest offered by the works we have just mentioned, the period of positive knowledge seems to us to date from Garrod's investigations in 1848. This observer, so often quoted in the course of these lectures, has established: *first*, that in acute or chronic gout there is an excess of uric acid in the blood: *secondly*, that from the first attack there is a deposition of urate of soda in the joints; *thirdly*, that during the attack or paroxysm there is an appreciable diminution in the excretion of uric acid by the kidneys.

These are the fundamental facts that may serve as elementary principles of a pathogenic doctrine; but there is still no physiological theory of gout in it. A few attempts, however, have been made in that direction, and I shall present you with the chief results thereof.

I.—The presence of an excess of uric acid in the blood does not constitute gout,[3] but merely induces a marked predisposition to this malady. We must, then, study the various circumstances that may augment the proportion of this excrementitious product; but we are met with difficulties at the very first step.

What is the origin, what are the sources of the uric acid excreted? Authorities do not agree concerning this point.

A.—The theory of direct combustion, advanced by Liebig, seems to afford a ready solution to the problem. Uric acid has its origin in the blood itself, at the expense of the albuminoid matters (fibrin, albumen, globulin) which have not been oxidized sufficiently to convert them into urea.

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[1] On Gout and Rheumatism, p. 3 and following. London, 1866.

[2] Andral: Précis d'Anatomie Pathologique. Vol. i., p. 533, and Vol. ii., p. 387. 1829. Rayer: Traité des Maladies des Reins. Vol. i., p. 243. Paris, 1839. Cruveilhier: Atlas d'Anat. Pathologique. Part 4, Plate iii.

[3] Albuminous nephritis and the saturnine cachexia are also among the number of diseases accompanied by an excess of uric acid in the blood.

Here is an excess of receipts over expenses; an individual has eaten too much, taken too little exercise, and hence, gout is developed.

But it has been recently proved that it is always the urea which increases, and not uric acid; and besides, according to the researches of Bischoff and Voigt, these two products are the result of the disassimilation of the elements composing the tissues, and are never DIRECTLY formed in the blood.

B. What, then, are these organs, what are these tissues from which uric acid is formed? Again we find ourselves in the face of contradictory results.

Urea comes from the muscles, we are told, and uric acid from the parenchymatous viscera; and, indeed, it has been found in brain and liver, in the spleen (Schérer), and in the lungs (Cloetta). A few pathological facts tend to confirm this idea: thus, Uhle and Ranke found an actual excess of this acid in the urine in a case of splenic leukæmia; and Harvey obtained the same result in diseases of the liver.

Other physiologists say that urates arise from cartilage and fibrous tissue, and organic movement is certainly less active in them because of their scanty vascular structure, as Bartels[1] has remarked; consequently, oxidation takes place in an incomplete manner. The investigations of Professor Robin[2] have led to an analogous result. He admits that in fibrous tissue the albuminoid materials are changed into glutin; and this substance, in its turn, breaks up into uric acid and urates by a process of disassimilation. Hence, it is evident, that when the work of disassimilation is exaggerated in these parts, the result will be a saturation of the blood with these products—in other words, a uric acid diathesis.

Professor Robin has also found uric acid in normal fibrous tissue;[3] and hence, the pathological condition is to him nothing but an exaggeration of what occurs in a state of health. He explains, in this way, why the articulations are the principal seat of the lesions in gout: their richness in fibrous tissue renders them unusually liable to be attacked in the disease.

Without denying the plausibility of this explanation, let us remark that the theory according to which uric acid and urea are the immediate products of disassimilation is certainly deserving of respect, although after all it is merely an hypothesis. Its foundation rests especially on the presence of these two substances in normal blood; but they are found only in the minutest proportion in mammals, while in birds and reptiles they seem wholly wanting.[4] The presence of urea and uric acid in the tissues has also been advanced in support of these ideas; but as for urea, this is not the fact, except in morbid states; in the normal condition of affairs kreatin and kreatinin alone are found in the muscles. Concerning uric acid, the *case* is better proven.

In any event, the investigations of some of the modern scientists seem to invalidate this hypothesis. According to Zalesky, urea and uric acid are formed in the kidney at the expense of kreatin: tie the ureters in a dog, and you will have an accumulation of urea in the blood; extirpate the kidneys, and there will be no such phenomenon. In reptiles there will be an accumulation, not of urea, but of uric acid, when the ureters are ligated;

[1] Deutsche Archiv für klin. Med., Bd. i., Heft 1, p. 13. Leipzig, 1865.

[2] Dictionnaire de Méd. (de Nysten), p. 678. 1865. Programme du Cours d'Histologie, p. 90. 1864.

[3] A verbal communication from Professor Robin.

[4] Zalesky: Untersuch. über den urämisch. Process. Tübingen, 1865. Recent researches of Gréhant appear to invalidate the results obtained by Zalesky.

but ablation of the kidneys is followed by no such occurrence. Zalesky concludes therefrom that urea (in those animals that secrete it) and uric acid are formed within the kidney itself, and have not previously existed in the blood.

In this direction, then, we do not find any really important data, any really solid foundation upon which we can establish a rational doctrine, and hence we must have recourse to other means of investigation.

II.—The purely experimental research into the conditions which induce a variation in the proportion of uric acid in the renal excretion at least furnishes us with some interesting information.

The proportion of uric acid increases after a meal (Bence-Jones); fasting diminishes it by one-half, and a vegetable diet acts in a similar manner.

All authors since Lehmann agree as to the effects of a purely animal regimen; there is an increase of uric acid and urea, but especially of the latter product.

Theoretical deductions seem to accord quite closely, up to this point, with the data furnished us by observation from the standpoint of gout's etiology. But in proceeding with this study we shall very soon encounter contradictions.

It is, for instance, a generally admitted fact that exercise is one of the best means for preventing the uric acid diathesis. Lehmann's experiments confirm this view; he has established beyond all dispute that muscular activity results in an augmentation of the quantity of urea and a diminution in the proportion of uric acid. These results have been confirmed in the case of urea, but contradicted concerning uric acid. Beneke, Genth, and Heller [1] found that prolonged exercise during three hours had the effect of increasing the quantity of this product. Ranke,[2] and Speck admit that unusual activity may be followed by the same results. And, in the main, however violent or prolonged the work may be, there is rather an increase than a diminution of uric acid.

Concerning the activity of the respiratory functions, it is generally admitted that the more this increases, the greater is the diminution in uric acid, while the proportion of urea is augmented. Still, it must be acknowledged that this view rests upon no very well established facts.[3]

In spite of the interesting experiments of Böcker,[4] we cannot as yet conclude anything concerning the influence of alcohol. According to this investigator, alcohol and spirituous liquors diminish the production of urea and urea acid; wine, on the other hand, tending to increase it, as the previous experiments of Liebig had already proved. When beer does not act as a diuretic, it diminishes the quantity of urea and increases that of uric acid, but only very slightly. Finally, tea and coffee diminish the proportion of this substance. If, in these experiments, it be admitted that the quantity of uric acid passed in the urine corresponds to that formed in the economy—which is quite probable, since the subjects who have aided in attaining these results were in full health—it can be proved that alcohol and spirituous beverages act here wholly different from beer and wine, agreeing thus with the data of clinical observation.

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[1] Beneke: Nord See Bad., p. 85, 1855. Genth: Untersuch. über den Einfluss der Wassertrinkens auf dem Stoffwechsel. Wiesbaden, 1856. Heller: Heller's Archiv Neue Folge.

[2] Ranke: Aussch. der Harnsaure. München, p. 240. 1858.

[3] Consult Parkes: On the Urine, pp. 50 and 220.

[4] Böcker: Beitrage zur Heilkunde. Vol. i., p. 240.

Upon the whole, contemporaneous chemical and physiological sciences have not yet shed a very brilliant light upon the all-important question in gout, namely, the presence of uric acid in the blood.

Since, however, the reality of the latter phenomenon has been experimentally proved, can we, accepting this as a starting-point, deduce from it the other symptoms of this disease? This has been the aim of Garrod's endeavors; and we shall exhibit the results of his labor, asking you to notice, however, that it is now no longer a deduction of a general theory of gout, but only the question of an attack.

III.—We have already seen that various circumstances are preparatory to the occurrence of a gouty attack and render it imminent. Some have the effect of accumulating uric acid in the blood, either by directly favoring the production of it (excessive meals and abuse of certain beverages), or by diminishing the excretion of this product (lead-poisoning, painful emotions). Others, again, attack the solubility of uric acid in the blood by diminishing the latter's alkalinity; such are the impression made by cold, checking the acid secretion of the sweat, and the use of acids, vinegar, etc.

We may suppose, then, that the presence of an excess of uric acid abruptly thrown into the circulatory stream explains the nervous derangements, the dyspeptic and other premonitory symptoms which immediately precede an attack of gout.

Up to a certain point the local symptoms may also be referred to a similar explanation. We shall now review the most important of these phenomena, assuming, for that purpose, a standpoint such as we have just described.

Gout preferably affects the articulations. Here is a point of similarity with other dyscrasiæ which have a predilection for the joints. We see this in purulent infection, in glanders, and in the therapeutical exhibition of arsenic; and lactic acid introduced into the veins seems also to expend its action upon the joints (Richardson).

Gout preferably affects fibrous tissue, and especially cartilage. This unpleasant prerogative may be attributed to their scanty vascular structure and to the relatively slightly alkaline reaction of their own tissue—two circumstances which clearly favor the formation of those crystalline deposits characteristic of the disease.

Gout preferably invades the metatarso-phalangeal articulation of the great toe. This perhaps arises from the fact that the joint is one of the farthest from the circulatory centre; and it also undoubtedly occurs because this articulation is often called upon to support the entire weight of the body, frequently presenting lesions before any gouty manifestations appear. It is well known, besides, that traumatic causes have the effect of inducing the invasion of gout.

We can explain, to a certain extent, the successive implication of the articulations, for when extensive deposits have formed upon the cartilages of a joint, it may be said that this point is saturated; whereupon the other articulations take it up, following a more or less regular order.

Tophi also acknowledge as their cause a saturation of the cartilages; and thus their formation is always a subsequent phenomenon.

It may be asked whether the deposits of urate of soda in the cartilages are the cause or the effect of the local inflammation. Garrod inclines toward the former view. He says that the inflammation excited by these deposits seems to have the effect of destroying the urate of soda, and that, following an attack, the blood contains considerably less of this salt. Be-

sides, the deposits which form externally are not preceded by inflammatory action, and, even if they sometimes give rise to symptoms of this nature, it is solely because they are foreign bodies (external ear).

And thus the formation of these deposits in the cartilages precedes the first attack, while the formation of new deposits, either in the same joint or in new articulations, brings about those local phenomena that characterize subsequent attacks.

But why this excruciating pain inaugurating the series of articular symptoms? It cannot be attributed to inflammation; local fluxion is precisely as intense, but certainly far less painful in articular rheumatism. According to Garrod, we must attribute it to the presence of the deposits themselves deep in the cartilages, and to the tension which they induce thereby; for only when gout is *intra-articular* is the suffering so great; when the deposits are external, it is not equally severe.

Finally, symptoms of arthritis appear, and a general reaction is caused by the local phenomena, it being well known that its intensity is usually in proportion to the number of articulations attacked and the degree of local inflammation.

Such, gentlemen, is the state of our knowledge upon this subject. I have considered it proper to devote myself to a discussion frequently barren, in order to show you how much progress, in this respect, there yet remains for us to make.

Concerning visceral gout, we have previously described the results of Zalesky's interesting experiments; these have shown us that the ligation of the ureters induces, in many animals, the formation of deposits of urate of soda in the stomach-follicles. It is highly probable that in man the gastro-intestinal juices are loaded with urates, in case of saturation. Undoubtedly, analagous phenomena might also be produced bearing on the other points. In those animals upon which he experimented, Zalesky found a large proportion of urate of soda in the muscle-extract. We all readily understand the importance of these facts from the standpoint of gout's visceral symptoms, but what is less easily explained are the sudden metastases which carry the morbid action from one point to another—from the great toe to the stomach, and from the stomach to the joints. Concerning this point, science has certainly not pronounced her last dictum.

It yet remains for me to speak of the therapeutical measures which we can employ against gout, but I prefer to leave this until we take up the treatment of chronic rheumatism; for, by thus bringing them together, we shall find the elements of a comparison as curious as it is instructive.

LECTURE XI.

CHRONIC ARTICULAR RHEUMATISM AND ITS ANATOMICAL LESIONS.

Summary.—Chronic Articular Rheumatism Essentially a Hospital Disease—Nature of the Malady—Relation to Acute Rheumatism—Principal Varieties of this Affection—Chronic Progressive Articular Rheumatism (Gouty Rheumatism)—Chronic Partial Articular Rheumatism—Heberden's Nodes; not to be confounded with Gout.

Anatomical Characteristics of Chronic Articular Rheumatism—Necessity of a Careful Study of the Local Lesions—Unity of the Disease—Earliest Works Relative to this Subject.

Fundamental Characteristics of Chronic Rheumatismal Arthritis—Changes in the Synovial Membrane; in the Articular Cartilages; in the Intra-articular Fluid; in Osseous Tissue—Histological Study of these Different Lesions—Modifications corresponding to the Chief Clinical Forms of the Disease.

GENTLEMEN:—After having thoroughly studied the history of gout, we are now about to take up a disease so closely allied with it, that very frequently the two conditions have been confounded; but we hope to be able to prove to you that this similitude is without foundation, and that chronic articular rheumatism has a place reserved for itself apart from gout.

With regard to chronic articular rheumatism, the succeeding subject of these lectures, we enjoy a great advantage; for, while gout is not one of the diseases commonly met with in the hospitals of France, and is, besides, an affection quite infrequent in the female sex—while gout is almost unknown in the Salpêtrière, chronic rheumatism is, on the contrary, one of the commonest infirmities in this institution; and indeed, this disease prevails among women and among the least-favored classes of society. In this way the proportion of patients admitted to the hospital for this kind of lesion is about one-fifteenth of the total number of inmates.

Most of those authors who have made a special study of this disease have drawn on institutions analogous to the Salpêtrière for their observations. In England the *workhouses* furnished the interesting materials for Colles, Smith, and Adams' publications. It was, you know, in the Salpêtrière that Landré-Beauvais wrote the monograph which we have already had occasion to quote. We are about to undertake, then, a truly clinical study, and I shall frequently have the opportunity to present you, not only with anatomical specimens, as has so often been done up to this time, but even with patients who are suffering the lesions I shall describe to you.

I.—The name I have chosen to designate the disease in question sanctions a nosological interpretation which I unreservedly maintain, but which all authors do no admit.

Among the adversaries of the opinion which I hold, some declare that we have here to do with a special disease, one completely independent of gout and acute articular rheumatism: this is Garrod's *rheumatoid arthritis* and the *rheumatic gout* of Fuller. Others again, consider the various forms of nodular rheumatism as subordinate to gout.

I shall endeavor, gentlemen, to justify the ideas which I uphold, and to show you that chronic articular rheumatism is *sometimes* seen, at the bedside, proceeding directly from the acute form, precisely as chronic lobar pneumonia may follow acute pneumonia. The fact is, however, that the chronic form of articular rheumatism almost always develops spontaneously, and without passing through the acute form ; but this negative fact cannot invalidate the connection we are endeavoring to establish.

As for gout, we shall institute, later on, a radical distinction between the two affections.

II.—Chronic articular rheumatism appears in various aspects, and of such dissimilar appearance that many authors have concluded that several different diseases are presented them. We, however, *only* see in them the various forms of one and the same affection.

To give but a single illustration, it is enough to state that many writers are ready to admit that nodular rheumatism is nothing but a poly-articular rheumatism in the chronic stage ; but they refuse to recognize a rheumatismal origin for the disease when it is localized in a single joint, and there slowly and insidiously produces the grave and deep-seated lesions of *morbus coxæ senilis*.

We hope to be able to prove to you that it is quite impossible to make an actual distinction between the various forms of rheumatism, but that, on the contrary, it is frequently possible to show that they all proceed from one and the same source.

It is, however, indispensable, from a clinical standpoint, to make a separate study of the principal varieties of chronic rheumatism, just as if it were really a question of several distinct diseases ; this is the only way to avoid confusion. And this preliminary work finished, we shall endeavor to demonstrate the common tie binding them together.

There are numerous types of chronic articular rheumatism ; but we shall devote our attention chiefly to the following :

1. *Chronic Progressive Articular Rheumatism.*

This is the *gouty* or *nodular rheumatism* of certain authors : the *primary asthenic gout* of Landrè-Beauvais, and Haygarth's *nodosities of joints*. This is the gravest form of the disease, being accompanied by deplorable infirmities. Although exhibiting a preference for the smaller joints, it may also invade the large ones ; and it frequently induces muscular retractions and other symptoms.

Two kinds may be distinguished : this disease, sometimes *primary*, sometimes *secondary* to the acute (the latter rare, however), may be either benign or malignant.

It is not always concentrated in the joints, but may be accompanied by visceral diseases, at times analogous to those of acute rheumatism (occasionally proceeding therefrom), and at others again by those peculiar to chronic rheumatism ; in the latter category we must place *ophthalmia* and *albuminous nephritis*.

The local changes are those of a *dry arthritis*, which are, besides, excepting a few secondary modifications, common to all the forms of chronic rheumatism.

2. *Partial Chronic Articular Rheumatism.*

The joints affected in this variety are few in number, sometimes only one. The articular changes are the same as in the preceding variety, but are much graver, as seen in the case of *morbus coxæ senilis.* Foreign bodies, which sometimes develop within the joint, are frequently, in such cases, of an exceptional size.

Visceral implication or abarticular affections are, on the contrary, quite infrequent here, although they sometimes do exist. It is in the benign form of the disease that we are especially liable to meet with them—certain forms of asthma, and skin diseases, occurring in some of its subjects. In the malignant form, where visceral affections are rare, and where the whole disease seems to be concentrated upon the affected joint, the case is wholly different, although albuminuria has been seen in a few exceptional cases.

3. *Heberden's Rheumatism.*

Heberden's *nodosities* (*digitorum nodi*) constitute the mildest type of this disease.

It is at this point especially that it becomes necessary to enter into a critical discussion. When it is a question of nodular rheumatism, the difference from gout, at least in certain characteristics, is generally conceded ; but it is not the same with the variety we are describing. It may be said that no one doubts but that these lesions are certainly and consistently related to gout. We are constrained to hold a diametrically opposite opinion ; and we purpose to describe in a very painstaking manner the arthritis which preferably attacks the *second articulation of the fingers,* deforming them in such a strange fashion.

These lesions have certainly engaged the attention of many an investigator, but they have not yet been studied with all the care they deserve. Pathological anatomy will very soon enable us to recognize that, excepting their special seat, these arthropathies differ in no way from those which constitute the two preceding types ; and we shall eventually have the opportunity to justify, from a clinical point of view, the separation of this particular type from them.

It is enough, at present, to observe that in ordinary cases we meet here only with nodosities of the terminal phalanges, almost always indolent and without any complication. Sometimes, however, there are several other joints, some of which are among the most important articulations, that are simultaneously affected ; and indeed, the articular lesion may be accompanied by muscular or neuralgic pains ; sometimes in the sciatic, the trifacial, or in other nerve-trunks.

Among the visceral diseases which may occur in this variety, we may especially indicate *asthma* and *megrim.*

III.—We are now about to consider the anatomical characteristics of chronic rheumatism.

When we attempted to sketch the history of gout, we met at the very commencement a fundamental fact which governed all the symptomatic manifestations of the disease—we mean the *change in the blood from an excess of uric acid.* We find no characteristic with such a general bearing to guide us in the study of chronic rheumatism ; and although it is probable

that a special modification in the fluids of the economy exists in rheumatism as well as in gout, the hypothesis is still far from having been demonstrated.

A thorough and careful examination of the local lesions is, therefore, what we must here have recourse to; and we shall pursue our researches from the dual standpoint of the clinic and pathological anatomy, though laying especial stress, for the time-being at least, upon the latter aspect of the question.

In the first place, pathological anatomy enables us to establish the unity of this disease; for the various forms it may assume are distinguished by clinical characteristics principally, presenting, with respect to local changes, a common type modified by a few differences of lesser import. In the second place, it enables us to unite the chronic with the acute or subacute form of articular rheumatism; and finally, to demonstrate a radical difference between this affection and those other diatheses, which, along with it, enjoy the privilege of localizing around the joints; such, for example, are gout, scrofula, and syphilis.

We are not compelled to go very far back in history to meet with the first works bearing upon our subject.

The physicians of antiquity, as we have already remarked, seem to have confounded articular rheumatism with gout, and we may ask ourselves whether the first of these two diseases is not one of those peculiar to modern times. Archæology comes to the aid of medicine on this question, and Pompeiian excavations tell us that chronic rheumatism was already in existence during the first century of the Christian era. Delle Chiaje, in a work entitled "Osteologia Pompeiana," has depicted articular lesions identical with those which are found in the plates of Adams' classical work. Concerning this point, then, there can be no doubt whatever; but chronic rheumatism was not recognized as a separate morbid species until the time of Sydenham and Musgrave, and the first monographs devoted to the study of this question date from the commencement of this century. We may quote in particular that of Landré-Beauvais (an VIII.), Haygarth (1809), and Chomel (1813). A little later on, the fundamental features of chronic rheumatismal arthritis were published; in France, Lobstein (1832), speaking with reference to *arthritic osteopsathyrose*[1] noticed that the bones in this disease were especially fragile, that destruction of the articular cartilages was followed by eburnification, and that osseous vegetations formed around their articular extremities. About the same time an Irish physician, Colles, remarked that this inflammation differed from others by a very especial characteristic; he says: "Two very different processes are taking place at the same time, namely: absorption of the old bone and its articular cartilage, and the formation of new osseous tissue."

But to Adams,[2] a contemporary and countryman of Colles, we are especially indebted for the best studies that have been made of this subject (1839–57). So far as examinations by means of the naked eye are concerned, his descriptions leave but little to be desired; but a special mention is due the works of Deville and Broca upon *dry arthritis* (1850). In certain respects they completed the descriptions given by Adams. In our days histological investigations have shed a penetrating light upon this question.

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[1] *Osteo-psathyrose:* a condition of bone accompanied by extreme fragility, and probably the *fragilitas osseum* of English, and the *osteoporosis* of German and some French authors.—L. H. H.

[2] A Treatise on Rheumatic Gout. By R. Adams, M.D., etc. London, 1857.

In Germany, Zeis, H. Meyer[1] and Otto Weber,[2] tell us how each tissue is changed under the influence of chronic rheumatism, and have in this way suggested reasons for many phenomena that otherwise would have remained unexplained.

In France, Ranvier, Cornil, and Vergely,[3] have confirmed and developed the results of these investigations.

It is by means of these documents, and the studies we ourselves have made, that we shall describe to you the morbid action that characterizes chronic articular rheumatism.

A.—Summary of the Fundamental Characteristics of Chronic Rheumatismal Arthritis.

The disease we are about to study involves all the structures that constitute the joint, but it is first exhibited upon the synovial membrane and the articular cartilages; these two parts may be affected simultaneously or in succession. The synovial membrane becomes extremely vascularized; the pre-existent synovial fringes increase in size and there is a formation of new villous appendages; and finally, foreign bodies may develop, either at the expense of the fringe-like processes themselves, or deep within the synovial membrane.

There is, at the same time, a change in the synovia of the articulation: at first there is an increase in the secretion of the fluid, and this, according to Adams, is a constant occurrence. Later, the intra-articular fluid may undergo various changes, but it never contains *pus*, except when certain complications exist. Destruction of the articular cartilage occurs in a manner already known to William Hunter, and thoroughly studied by Redfern;[4] it passes under the name of the "velvety" change.[5] The cartilages are first seen to break up into a fibrillar structure, then these fibrils themselves disappear, and the cartilage is destroyed.

Now, let us see what are the changes occurring at the same time in the osseous tissue. There is, first, eburnation of the articular surface, either at the expense of the deeper portions of the cartilage or of the pre-existing bone; osseous vegetations simultaneously occur, located, most commonly, at the extremities of the cartilage. These osteophytes have, at first, a cartilaginous structure, but soon are impregnated with calcareous salts, finally ossifying.

A third change, whose importance at least equals that of the preceding lesions, is the rarefaction, the decrease in density, of the osseous tissue at the articular extremities. At *first* there is marked vascularization beneath the layer of ivory-like substance, and a bony marrow of new formation develops at this point; *secondly*, the bone becomes very much less dense, and is transformed into a kind of fatty marrow underneath this spot.

These are the fundamental facts which it is important to state here. There are, no doubt, many other alterations; but, since they are not common to all the forms of articular rheumatism, we shall reserve their study for a more suitable occasion.

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[1] Müller's Archiv. 1849. [2] Virchow's Archiv, p. 74. January, 1858.

[3] Ranvier: Thèses de Paris. 1865. Cornil (translation of Niemeyer: Pathol. Interne. Vol. ii., p. 556. Vergely: Thèses de Paris. 1856.

[4] Edinburgh Monthly Journal. 1849.

[5] The numerous fibrillæ at right angles to the articular surface give to the latter a velvety appearance.—L. H. H.

At present we shall take up each point just described, and study it in a more thorough manner, aided by all the means of investigation which to-day are at our command.

B.—Histological Study.

I.—*Changes in the synovial membrane.*—To a certain extent, these alterations merely consist in an exaggeration of the condition existing normally in a rudimentary state.

It is well known that the synovial membrane has fringe-like processes which themselves present appendages.[1] In the pathological state these little prolongations are seen increased in number, and presenting a greater vascularity.

Normally, there are cartilage-cells in these synovial appendages (Kölliker). These cartilaginous nuclei may become the starting-points for those pediculated foreign bodies of which we have already spoken. According to Ranvier, there is, first, cell-proliferation; then the formation of true cartilage; calcification next takes place; and finally we have ossification properly so-called, with bone-corpuscles.

Within the deeper structure of the synovial membrane *sessile* bodies may be developed, passing through precisely the same phases as those which are pediculated.

2. *Changes in the cartilage.*—With regard to articular cartilage, we find ourselves confronted by two principal facts: the first is *proliferation* of cells, and the formation of secondary capsules; the second is *segmentation* of the fundamental tissue. It breaks up into fibrils which are free at that extremity corresponding to the articular cavity.

Fig. 12.—Nodular rheumatism: surface of a cartilage from a phalangeal articulation. (After Ranvier). *a*, primary, filled with secondary capsules; *b*, segmented fundamental substance. *The primitive capsules have just emptied their contents upon the free surface of the cartilage.*

This pathological process ought to be studied on the surface of the cartilage, and in its deeper parts.

Upon the surface this segmentation has the effect of opening a passage for the capsules of the cells, and then the contents of these capsules escape into the articular cavity. They are frequently found along with a *débris* of epithelial cells (Rindfleisch, O. Weber), and at other times they undergo colloid metamorphosis.

Concerning the fibrils of the cartilage's fundamental substance, they undergo mucous degeneration or softening, and are transformed into mucosin (Rindfleisch), which is discovered in considerable quantities in the articular fluid.

Again, those portions of the cartilage thus altered are very gradually worn away by articular rubbing, finally dropping off and laying bare the bony surfaces.

Within *the deeper parts* cell-proliferation results in the formation of a new bony layer; the primitive capsules become infiltrated with calcareous matter and open into the superficial medullary spaces; the cells which they

[1] Kölliker: Eléments d'Histologie Humaine, trans. of Béclard and Sée, p. 253. Paris, 1856.

contain become the embryonic cells of the marrow, and at their expense there is formation of new osseous tissue.

In this way eburnation of the surface occurs; it is a sort of sclerosis of the bone, accompanied by vascularization of the deep parts. Here we notice a strange phenomenon taking place, and one recalling, in a certain rough way, the facts observed by geologists relative to the action of glaciers upon rocks. The ivory-like surfaces present striæ—radiating lines that are of greater or less depth in the direction of articular movements, and thus evidence an imperfect repair in the presence of the wear ("usur") occasioned by the constant rubbing of the surfaces.

The articular cartilage, as you know, is covered at its peripheral portion with synovial membrane. According to Ranvier, this condition, in cases where the articulation is invaded by rheumatism, hinders the capsules opening into the articular cavity and pouring their contents into it. But they continue, then, to proliferate at the spot, thus determining the formation of those rounded swellings or pads which are noticed on the part, and which, at first cartilaginous, ultimately become osseous.

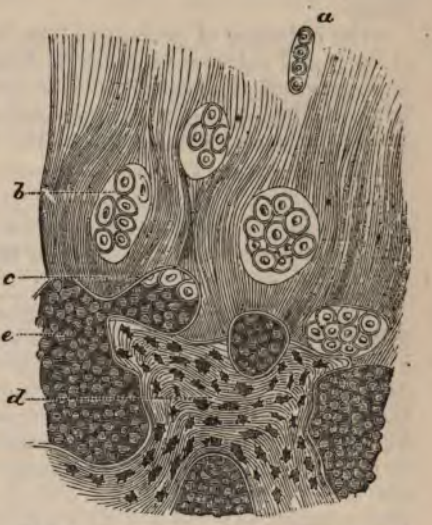

FIG. 13.—Nodular rheumatism: deep layer of a phalangeal cartilage. (After Ranvier.) *a*, normal capsule; *b*, capsules near bones, enlarged and filled with secondary capsules; *c*, primitive capsules about to open into the medullary spaces and to empty their contents therein; *d*, osseous substance; *e*, marrow filling the areolæ upon the surface. At this point it is embryonic, for the capsules have poured out their contents there; a little farther on it is adipose.

Ranvier also ascribes all the new bony formations developed under such circumstances in cell-proliferation of the articular cartilage. The periosteum, however, probably assumes a certain share in their production, while there may likewise be simultaneous ossification of the articular capsules, the ligaments, tendons, and muscles. As for the interarticular ligaments—the meniscuses, etc., they are worn down and made to disappear by a mechanism analagous to that which destroys the cartilages—a subject which, however, has not yet been sufficiently studied.

These, gentlemen, are the most general facts that I find necessary to describe to you; but there are numerous modifications in this respect, according to the clinical form of rheumatism under consideration, and to some particular circumstances resulting from the very conditions of the disease. It is in this way that the changes met with in an articulation enforced to absolute repose differ from those accompanying more or less complete preservation of movement. We have proceeded until now, under the latter hypothesis, but at present we shall study the changes arising in cases where the joint is immovable.

In such instances, according to Adams, eburnation is no longer observed, but there is a neoplasia of connective tissue at the expense of the synovial membrane. According to Forster, the cartilage may also participate in this process; sometimes the fundamental substance may undergo

this change; and again, on the other hand, it may be the cartilage-cells that assume the appearance of connective-tissue cells. Whatever may be the value of this theory, we see the formation of embryo-plastic tissue uniting the bones together, and, at a certain period, becoming vascularized; then an ankylosis is formed, which is sometimes fibrous, sometimes osseous. The latter occurrence is very rare, taking place only in the very small joints.

The influence of prolonged rest has, moreover, the effect of leading to atrophy and extreme friability of bone-tissue; and perhaps there is a disappearance of the round, pad-like swellings with the corresponding vegetations. This atrophic process, common in general rheumatism, is, likewise, sometimes seen, though rarely, in *morbus coxæ senilis* (Adams).

C.—Modifications Corresponding to the Principal Forms of Chronic Articular Rheumatism.

The description we have just given applies principally to general and to Heberden's rheumatism. Here we find the changes of a dry arthritis in a rudimentary condition; but in partial arthritis they present an enormous development, and become almost unrecognizable. Immense osseous vegetations are seen to form: as, for example, in *morbus coxæ senilis*. Wearing erosion ("*usur*,") and eburnation of cartilage and bone are exhibited in the highest degree, and eventually result in a deformity of the head of the bones. And finally, it is here that atrophy of bone is manifest to the very greatest extent—lesions which used to be explained by osteomalakia and senile rachitis (Malgaigne, Hattier).

In those articulations which, for reasons still but imperfectly known, admit of the presence of foreign bodies, an immeasurable quantity of these is seen developing; as, for example, in the shoulder and knee; but it is quite the contrary in the case of the hip-joint, or the joints of the fingers. There is, besides, considerable thickening of the fibrous capsules, and ossification of ligaments and tendons. Let me remark, however, that these differences cannot justify a radical separation; in partial rheumatism there is a large number of joints where changes have occurred only in a very slight degree, and in general rheumatism some of the diseased articulations present lesions quite as marked as in the partial variety; this, for example, occurs in the vertebral column.

To complete this study we must needs institute a comparison, with reference to their anatomical points, between chronic rheumatism and the other arthropathies of slow development. But, before broaching this subject, we desire to point out the analogies which bind the chronic to the acute form of articular rheumatism.

LECTURE XII.

COMPARISON BETWEEN CHRONIC ARTICULAR RHEUMATISM AND THE OTHER CONSTITUTIONAL ARTHROPATHIES, FROM AN ANATOMICAL STANDPOINT.

Summary.—Analogy between the Lesions of Chronic Articular Rheumatism and those of Acute—Changes in the Joints in Acute and Subacute Articular Rheumatism—Sometimes Null and Insignificant, sometimes Manifest—Arthritis with Exudation—The Inflammation not Superficial—The Cartilages and Bone may participate in the Process—Lesions of the Synovial Membrane—Lesions of the Articular Cartilages—Lesions of Bone—Nature of the Fluid poured out into the Synovial Cavity—Analogy between these Lesions and those of Chronic Rheumatism.

Characteristics that Distinguish Arthritis Deformans from other Arthropathies—Arthritis from Prolonged Repose—Scrofulous Arthritis—Syphilitic Arthropathies—Gouty Diseases of Joints.

The Changes in Chronic Rheumatism Lack a Specific Character—They may arise from Many Causes Foreign to Rheumatism—They are then almost always Mono-articular—Chronic Rheumatism, in the Majority of Cases, a Poly-articular Disease.

GENTLEMEN:—We are now in a position to compare, from a pathologico-anatomical standpoint, chronic articular rheumatism with other diseases of joints which develop slowly.

But, before we undertake such a comparison, it is indispensable that we should bring out the points of similarity existing between the changes in chronic articular rheumatism and those of the acute form. We shall very soon discover that the lesions of chronic rheumatism are, as it were, but a higher expression of those which occur in acute rheumatism; they correspond to a more advanced period of the morbid action.

The analogy we have pointed out is not, at the first glance, a striking one, especially if we compare extreme cases, as for example, flying articular rheumatism and *morbus coxæ senilis;* but it is manifested, on the other hand, most unmistakably when we choose for comparison those subacute cases that form, from a clinical as well as from an anatomical standpoint, a transition between the acute and the chronic form of articular rheumatism.

I.—CHANGES IN THE JOINTS IN ACUTE AND SUBACUTE RHEUMATISM.

In the scholastic language of ancient medical writers, the term *subacute* was applied to acute diseases when they endured more than twenty-one days—this being the extreme limit to acute affections properly so-called—and they might extend over forty days. Pathology does not tolerate such arbitrary divisions, and, under the name of subacute articular rheumatism, we shall describe a disease whose development is, in truth, slower than that of acute articular rheumatism, and, moreover, differs from it in other respects, without, however, being radically removed therefrom. But, it

must also be noticed, this subacute form is already allied to chronic articular rheumatism because of certain of its characteristics.

Thus, in this variety of the disease, the articular affections are more stable: fever is less intense, and resembles hectic fever; the smaller joints are frequently affected, and oftentimes a large number of them. It is well known that in acute articular rheumatism the case is just the opposite. And, finally, visceral diseases, or at least certain classes of them, as endocarditis and pericarditis, are here much less frequently observed.

Such are the fundamental characteristics of subacute articular rheumatism. Later on in the course we shall present you with other considerations bearing on this subject; but, for the time-being, we wish to indicate the lesions which are found in the joints in acute articular rheumatism, and also in the subacute form of this malady.

There were interminable discussions, at a certain epoch, concerning the lesions of acute articular rheumatism. Can, or cannot this disease terminate in suppuration? Some affirm that articular rheumatism never leaves a trace in the joints; and others assure us that it produces most serious lesions, which may induce a purulent arthritis.

To-day the question is considered to have been considerably exaggerated on both sides, and a sounder appreciation of facts has resulted in establishing the truth of this opinion.

True rheumatismal inflammation of the joints sometimes leaves behind it no appreciable change (Grisolle, Macleod, Fuller), although, in certain cases, on the other hand, it may result in a purulent arthritis (Bouillard). But examples of this kind are very exceptional, and what we meet with in the vast majority of cases are the characteristics of an arthritis accompanied by a sero-fibrinous exudation: the synovial membrane is red and vascular, and its cavity contains a serous fluid in which fibrinous floccules float.

For a long time it was thought that this was a completely superficial inflammation, and that the synovial membrane alone was implicated (synovitis); but to-day it has been conclusively established that the cartilages, and even the bone, may participate in these changes.

The synovial lesions will not long claim our attention: we have, *first*, a more or less pronounced vascularity of the fringe-like processes of the synovial membrane, which are present in the normal condition of affairs; and, *secondly*, a varicose dilatation of their vessels (Lebert).

The changes occurring in the cartilages possess a much greater importance; we are indebted for our knowledge of them to the interesting work of Ollivier and Ranvier.

It has already been observed (Garrod) that the articular cartilages acquire sometimes a certain opacity, and lose their polished appearance, their blue color, and the consistence which characterizes them in a healthy condition. Ollivier and Ranvier have shown, besides, that frequently there are changes which can be appreciated by the unaided eye; such are the partial swellings on cartilage that give it a mammillated appearance, and sometimes even cause actual erosion. But, in cases where there is no change visible to the naked eye, the microscope yet reveals palpable and probably constant lesions.

In the primary stage the most superficial chondroblasts become globular, and the cell which they contain divides, producing one or two secondary cells.

Thenceforth no alteration may occur in this condition, and we readily understand how, in such cases, the histological elements may return to their normal state; but in a more advanced stage, there is segmentation of the

fundamental substance in the horizontal direction—a kind of "velvety" condition marked by little furrows which penetrate more or less deeply into the tissue. In the interior of these little grooves of new-formation the capsules open and discharge therein the cells they contain; these too are

FIG. 14.

FIG. 15.

FIG. 14.—Acute articular rheumatism: surface of a cartilage from the condyles of the femur. (After Ranvier.) The fundamental substance is transversely segmented; the superficial capsules contain several secondary capsules; only one remaining normal, enclosing a small cellular mass.

FIG. 15.—Acute articular rheumatism: surface of the investing cartilage of the patella. (After Ranvier.) The primary capsules of this surface contain several secondary capsules; the fundamental substance is transversely segmented; an oblique strip, raised from the surface, remains floating.

then seen to mingle with the synovial fluid, therein to undergo mucous metamorphosis. In this case there are, as you see, striking analogies with what occurs in arthritis deformans.

But the changes which the bony surfaces present render the analogy yet more striking; in certain cases they seem to take part in the phlegmasic action. According to Gurlt,[1] the marrow of the osseous extremities undergoes decided vascularization attended with cell-proliferation. Hasse[2] and Kussmaul[3] have also alluded to some alterations in the bone and periosteum occurring in acute articular rheumatism.

A word yet remains to be said concerning the fluid contained in the synovial cavity. Sometimes it has an acid reaction, holding mucosin and albumen in solution; fibrinous floccules are seen floating in it, along with clots of concrete mucus and globular bodies, some of which are cartilage-cells or epithelial cells that have undergone fatty degeneration, while others greatly resemble pus-globules; and indeed, we occasionally find true pus-corpuscles in it. In general, however, it may be said that the latter element does not predominate, unless in those *exceptional* cases where there is symptomatic secondary rheumatism, or a rheumatismal arthropathy combined with the purulent diathesis.

In fine, if we desire to interpret these phenomena, fibrin and pus-globules correspond to an acute synovitis; while mucosin is produced by the transformation of epithelial cells and the fundamental substance of the cartilage.

These changes, presenting unquestionable analogies with those of chronic articular rheumatism, are yet more strikingly similar in the case of the subacute variety. Thus, in instances where rheumatism had lasted nearly two months, I have found thickening of the synovial membrane along with distinctly marked villous prolongations; while erosions and a decided "velvety" change were met with at several points.

On a subject who, on the twenty-fifth day, died after exhibiting cere-

[1] Forster: Handbuch der path. Anat., p. 1000.
[2] Hasse: Zeitschrift für rat. Med. Bd. V., p. 199–212.
[3] Kussmaul: Arch. für physiol. Heilkunde. Vol. xi. 1852.

bral symptoms, Bonnet found similar lesions even then.[1] Vestiges of these alterations are still found in those who succumb to organic diseases of the heart, after having previously had several attacks of acute articular rheumatism. There is an imperceptible transition between these cases and those where the disease has become decidedly chronic, on account of the prolongation or the incessant return of the rheumatismal affection. Then there are more profound changes: synovial villosities are developed, foreign bodies are beginning to be formed, the articular surfaces are commencing to undergo eburnation, and there is a formation of bony vegetations around the joint; finally the bone-tissue becomes friable toward the articular extremities.

Thus it is that we are enabled to prove, from an anatomical standpoint, the close connection binding the various forms of articular rheumatism among themselves. They are not different diseases; they are varieties of one and the same morbid species.

II.—Changes in the Joints in Certain Diseases, Independent of Rheumatism.

And now a word with regard to the characteristics distinguishing arthritis deformans from other chronic arthropathies.

First.—Arthritis from prolonged rest.—In the first stage, according to Tessier and Bonnet,[2] arthritis from prolonged repose gives rise to the following lesions: there is a sero-sanguineous effusion into the joint, even fluid blood sometimes being found there; the synovial membrane is injected, ecchymotic, and it is said there is sometimes even ulceration of the cartilages.

But in a more advanced stage I have found that central ulcerations cut as cleanly as with a punch, and peripheral ulcerations existed upon the articular cartilages. There are no osseous swellings, no fibrous ankyloses, but there is a decided rarefaction of the bone-tissue. But the essential characteristic of this arthropathy is the existence of a layer of connective tissue covering the articular cartilage throughout its whole extent; this membrane is readily detached from the subjacent surface, and we then find cartilage whose cells in the chondroblasts have undergone fatty degeneration. This membrane is frequently penetrated by arborescent vascularizations, sometimes advancing toward its central portions; in this way are probably explained the vascularizations of cartilage described by certain writers. I have found these lesions in patients who have long been stricken with hemiplegia or paraplegia, especially in those parts which remained uncovered, when articular deformity existed.

Certain of these changes, you know, are found combined with those of arthritis deformans in cases where the joint is absolutely at rest. Finally, we must acknowledge this to be an important question, one which, having as yet been little studied, demands further investigation.

Second.—Fungoid, or scrofulous arthritis.—The investigations of Ranvier[3] establish the fact that fungoid arthritis is widely separated from dry

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[1] Bonnet: Traité des Maladies des Articulations. Vol. i., p. 329. Paris, 1845.

[2] Tessier: Mémoire sur les Effets de l'Immobilité Longtemps Prolongée des Articulations. Lyon, 1844. Bonnet: Op. cit., vol. i.

[3] Ranvier: Des Altérations Histologiques du Cartilage dans les Tumeurs Blanches. Paris, 1866.

arthritis, even from a standpoint of elementary lesions. And this is proved by the study of the characteristic lesions of this disease. With regard to the bones and cartilages in arthritis deformans, there is a proliferation of the cell-elements ; in fungous arthritis, on the contrary, these elements are destroyed, and undergo fatty degeneration just as we see it occur in bone and in cartilage-cells. Besides, in a more advanced stage of the disease, the distinction is shown in the clearest possible manner ; fungoid arthritis gives rise to vegetations of bone and synovial membranes, attended by destruction and reabsorption of the cartilages ; and then supervenes caries or necrosis of the bone—peripheral abscesses, which surround the diseased joint, finally being formed.

There are, nevertheless, analogies between these two affections. In certain cases of scrofulous arthritis there is active proliferation of the cartilage-elements ; but this is a secondary occurrence. Sometimes there are

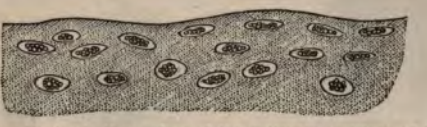

FIG. 16.—*A section perpendicular to the surface of the articular cartilage of the femur : from a child with a white swelling.* (After Ranvier.) The capsules at the surface each contain a little mass consisting of granulations that have accumulated within the cellular corpuscles. This is the change the cartilage undergoes at the commencement of the disease.

also produced osseous stalactites, but these are extremely vascular (Billroth) ; there is a very decided difference between them and the thick, blunt-edged stalactites, formed like drops of candle-grease, and usually but slightly vascular, which characterize arthritis deformans. In this way the elementary lesions of the two diseases differ from each other, although there are mixed cases in which the two may be combined.

Third.—Syphilitic arthropathies.—We now pass to the consideration of syphilitic diseases of the joints. It is extremely probable that by this denomination acute or chronic rheumatism occurring in the syphilitic has been described more than once, for these two diatheses are far from excluding each other. A few clinical peculiarities, however, and the decisive influence of specific treatment in certain cases where the so-called rheumatism has been protracted, have induced some physicians, among them Babington, Boyer, and Lancereaux,[1] to think that there really exist special arthropathies as the direct results of a venereal infection.

Lancereaux, to whom we owe a thorough and thoughtful work upon this subject, has carefully described the articular lesions of syphilis. He distinguishes two forms : (a) *secondary arthropathies*, which resemble acute or subacute articular rheumatism ; and (b) *tertiary arthropathies*, simulating certain forms of chronic rheumatism ; the latter were the only ones he was enabled to study from an anatomical standpoint. They commence in the subsynovial cellular tissue and in the fibrous tissue, being characterized by the formation of a neoplasm, which from its texture and external appearance absolutely resembles the gummy tumors (*gummata*).

In those cases recorded by Lancereaux there was no alteration in the synovial membrane, although the articular cartilages were eroded.

Fourth.—Gouty arthropathies.—The lesions of arthritis deformans frequently offer enough resemblance to those of gout to cause them to be easily confounded at the bedside ; but from an anatomical point of view

[1] Lancereaux : Traité de la Syphilis, p. 182. 1866.

this is not the case. You never find the least trace of a deposit of urate of soda, either in the articular diseases which arise from rheumatism, or in the other arthropathies we have just enumerated.

This uratic infiltration of cartilage is, then, the essential characteristic of articular gout; and besides, there is here no other constant lesion of the cartilage. There is no segmentation of the fundamental substance, and there is no cell-proliferation, so that if the two changes are found existing simultaneously there is evidently a juxtaposition of the two diseases. They never undergo a transformation that permits of their being confounded.[1]

FIG. 17.

FIG. 18.

FIG. 17.—*Section perpendicular to the articular surface: from a gouty cartilage.* (After Cornil.) *p*, articular surface; *v*, *n*, group of cartilaginous cells infiltrated and bristling with silky crystals of urate of soda; *o*, normal cartilaginous capsule, in contact with crystals formed in the fundamental substance of the cartilage. (Magnified two hundred diameters.)

FIG. 18.—A partly schematic representation of the dissolution of urates encrusting a cartilaginous cell by the action of acetic acid. (After Cornil.) *c*, cartilaginous capsules bristling with free crystals; *c'*, same capsule—the crystals are dissolved and other crystals of uric acid are forming; *c''*, capsule whose membrane is visible, while the cell yet remains incrustated; *c'''*, the whole cell clear, save only a small central nucleus.

The articular lesions of gout may be considered as the results of the presence of a foreign substance in the interior of the tissues; whereas the lesions of rheumatism, on the contrary, correspond to actual changes in the histological elements. And thus, when a piece of cartilage removed from the articular surface of a joint invaded by gout is treated with acetic acid, the crystals of urate of soda that infiltrate the cells are seen to dissolve, and these cells thereupon assume their normal appearance.

We think, then, that we have established, *first*, the unity of the various clinical forms of chronic articular rheumatism from a standpoint of morbid anatomy; *secondly*, the existence of an unquestionable relationship between the changes in acute arthro-rheumatism and those in partial or chronic arthro-rheumatism; *thirdly*, a decided difference, always considered from an anatomical point of view, between the disease under discussion and the other diseases of joints.

During this discussion, gentlemen, the question has no doubt presented itself to you: have the changes that we have just been describing any spe-

[1] Charcot and Cornil: Contributions à l'Étude des Altérations Anatomiques de la Goutte, in the Mémoires de la Société de Biologie. 1864.

cific nature? In other words, do they belong exclusively to the rheumatic diathesis?

Without adverting to anything which does not immediately concern chronic articular rheumatism, we are led to admit, even from a purely anatomical standpoint, the existence of sharply marked distinctions between the articular lesions this disease induces, and those which arise from gout, scrofula, and syphilis.

It must, nevertheless, be acknowledged that the elementary lesions which collectively constitute arthritis deformans may likewise be encountered in cases where a rheumatic cause cannot possibly be adduced. Thus, in *fungous arthritis*, proliferation of the capsules and segmentation of the fundamental tissue of the cartilage may here and there occur under the influence of an inflammatory action which, at a given moment, seizes upon various tissues. The rarefaction and condensation of bone-tissue at the articular extremities, the formation of osteophytes and osseous swellings around the articular cartilages, are, as you know, met with in those joint-diseases foreign to rheumatism. But it is the simultaneous change of the various parts in a joint, according to the method I have pointed out, and the extent which these lesions may acquire without being complicated by suppuration, that constitute, in our eyes, the *anatomical characteristic* of this disease.

But here we are led to ask whether an irritation completely independent of the internal cause that induces rheumatism—a blow, a fall, for example—can produce the various changes of arthritis deformans in a joint.

Certainly, gentlemen, we can recognize in these conditions all the lesions I have just enumerated; but in such cases they are almost always confined to a single articulation. But even here are we not authorized in intervening a latent cause that decides the nature of the local disease? Please to recall in this connection what occurs in subjects who are attacked with gout.

When articular affections are multiple—and this is most frequently the case—they always develop spontaneously, and seem to argue from this double claim a general predisposition of the economy.

In such cases the influence of *rheumatism* should be adduced; for in the present condition of science, we know of no other diathetic state to which we can attribute similar effects.

LECTURE XIII.

ACUTE ARTICULAR RHEUMATISM CONSIDERED ESPECIALLY IN ITS RELATIONS WITH CHRONIC ARTICULAR RHEUMATISM AND GOUT.

Summary.—A Succinct Description of Acute and Subacute Articular Rheumatism—Analogies with Chronic Rheumatism—Differences Separating it from Gout—Acute Rheumatism—Subacute Rheumatism.

Multiple Arthropathies—Pain—Swelling—Redness—Temperature—Duration—Variations in the Disease.

The General Constitutional Condition in Rheumatism—Fever—Irregular Progress of the Disease—Relationship between Intensity of Febrile Movement and the Number of Joints Affected—Pulse—Secretions—Saliva—Urine—Intense Anæmia.

Comparison between Acute Articular Rheumatism, Gout, and Subacute Articular Rheumatism—Pathological Blood-Conditions in Acute and Subacute Articular Rheumatism.

GENTLEMEN:—I do not intend to give you a complete description of acute articular rheumatism; such a study can only be pursued with advantage in the ordinary hospitals. Nothing, in reality, is rarer among old persons than the acute form of articular rheumatism; nothing, on the other hand, more frequent than the chronic form of this disease, as we have already proved to you.

Nevertheless, we cannot wholly overlook the history of acute articular rheumatism. We wish, in short, to maintain in the domain of the clinic the comparison we have established between these two diseases, when anatomically considered; and in thus taking this new point of view, we shall again establish the fact that not only do analogies exist between them, but that there is actual identity in certain of their points. In a word, we desire to complete the demonstration of the view we espouse, namely: that this is not a question of two fundamentally distinct diseases, such as certain authors try to demonstrate, but only of two different manifestations of one and the same diathesis; and you will easily recognize the salient points of resemblance uniting them, in spite of the diversities that result from the slow or the acute reaction of the organism in its symptomatic expression of disease.

But, on the other hand, we shall arrive at a conclusion just the reverse concerning the relationship of rheumatism to gout; we shall show you that if certain appearances sometimes bring these two diseases in proximity, this occurs only in very exceptional cases; and that these two diseases can almost always be clearly distinguished (thanks to the rules for diagnosis, which will be given farther on).

I shall now give you a concise description of acute articular rheumatism wherein we shall be careful to dwell only upon the fundamental characteristics.

I.—Description of Acute Articular Rheumatism.

We here find ourselves confronted by the two forms whose existence has already been pointed out: on the one hand, acute articular rheumatism, or the *rheumatic fever* of English writers; and on the other, subacute articular rheumatism (Garrod, Copland), or capsular rheumatism (MacLeod). Here there is a transition form, as we have already remarked, the characteristics whereof will later on be the subject of our study. At present we shall devote our attention to *rheumatic fever*.

The most frequently multiple arthropathies which we already know from an anatomical standpoint, and which we shall soon study under a clinical aspect, are far from exclusively constituting acute articular rheumatism. To them is joined a collective reaction, expressed by a most characteristic general condition of body, by a distinctly marked change in the constituents of the blood (increase in the amount of fibrin and diminution of the red blood-corpuscles), and by the frequent, we might almost say habitual, coexistence of certain visceral diseases. Endocarditis and pericarditis, for example, constitute one of the most fundamental occurrences in acute articular rheumatism; and this characteristic contributes, in great measure, to our regarding this affection as a general disease, or at least as one having no definite seat, and not simply as a collection of arthritises more or less independent of one another.

We shall arrive at the same conclusion by a study of the development of the disease among the etiological conditions that may give rise to it, among which heredity, as you well know, plays so manifest a part.

These, gentlemen, are the most general characteristics of rheumatic fever. Now, however, we must enter into some details bearing upon each of the points we have just described.

A.—*Arthropathies in Acute Articular Rheumatism.*

The characteristics of these local lesions are already known to you; I shall therefore confine myself to the elucidation of the analogies and the differences which approximate or distinguish them from gouty diseases of joints. We shall first study them separately, and later on regard them collectively.

First.—Pain.—This is pre-eminently nocturnal; its intensity is much less than in gout, but it is accompanied by muscular cramps, as in the case of the latter disease.

Second.—Swelling.—Tumefaction especially occurs in case of joints near the surface; it may have its seat in the adjacent cellular tissue, or result from distention of the synovial pouch by a serous or sero-fibrinous exudation. Contrary to what occurs in gout, there is no accompanying local œdema rendering it capable of pitting on pressure of the finger; Garrod, however, has sometimes seen this symptom occurring in cachectic individuals. There is no desquamation when the swelling subsides.

Third.—Redness.—There is an erysipelatous appearance. It is not so strongly marked as in gout, and presents no ecchymoses; moreover, the veins are less prominent.

Fourth.—Temperature.—According to Bouillard and Neumann,[1] there

[1] Neumann: Ergebniss und Studien aus der medicinischen Klinik zu Bonn, p. 33, Leipzig, 1860.

is sometimes a difference of a degree Centigrade (nearly one and one-half, Fahrenheit) in the temperature of the affected locality and the surrounding parts which do not partake of the heat resulting from the pathological activity.

Fifth.—Duration.—Each one of these cases of arthritis lasts, according to Budd,[1] about from three to fifteen days.

Let us now consider articular affections in their mutual relationships.

Generally, several joints are attacked at the same time. A rheumatism mono-articular from the commencement probably does not exist; when it is secondary, it is rheumatism located in a single joint. We will admit, however, that rheumatism may be partial; that is to say, it may have its seat in a small number of joints, in contradistinction to that which is general or poly-articular. In acute gout, you know, the disease is rarely seen as a general one from the beginning.

As for its mode of invasion, there are characteristics that sharply differentiate rheumatism from gout. According to the researches of Professor Monneret,[2] when rheumatism is mono-articular, it almost never affects the great toe; besides, the disease simultaneously invades the upper and lower extremities. In the majority of cases the knee, wrist, elbow, and instep are its places of election. Rarely are the smaller joints affected, except in the case of subacute rheumatism.

According to Budd and Professor Monneret, it is the tibio-tarsal articulation that is most frequently the first to suffer invasion.

Rheumatic arthritis is often developed symmetrically upon both sides; but too much importance must not be accorded to this occurrence, since it is nearly a common characteristic of all diathetic arthropathies.

Sometimes the abrupt disappearance of articular symptoms coincides with the sudden development of a visceral affection. But this is the exception rather than the rule; in any case, here is no experimental proof of a *retrocession* of the disease induced by external causes, as we have seen was the case in gout.

Finally, one of the most essential clinical characteristics of this disease is its excessive instability, permitting it to dart from one articulation to another, and frequently to change its seat during the course of the malady.

B.—*The General—Constitutional—Condition in Acute Articular Rheumatism.*

Here the fundamental occurrence is *fever;* and, indeed, febrile reaction, manifested by a more or less marked rise in the general temperature, is never wholly wanting during the course of acute articular rheumatism.

The heat of internal parts may exceed 40° C. (104° Fahr.), but it usually remains between 39° and 40° C. (102° and 104° Fahr.), according to Wunderlich, Hardy, and Sidney Ringer.[3]

The febrile movement assumes the continued type, with exacerbations and remissions which are generally very distinctly marked. The thermometric curves are here very irregular, and, according to Wunderlich, fail to furnish us with any exact information relative to the progress of the

[1] Tweedie's Library of Medicine, Art. Rheumatism. Vol. v., p. 191.

[2] Monneret: Thèse de Concours pour le Professorat, p. 51. 1851.

[3] Aitken: Science and Practice of Medicine. Vol. ii. Reynolds: A System of Medicine. Vol. i., p. 896.

disease; but what is most frequently observed is the maximum temperature during the day, and the minimum during the night.[1]

This disease, then, does not progress according to any regular type, it presents no *cyclic* course; there are no stages succeeding one another at definite intervals, as in pneumonia or the eruptive fevers; we do not have a severe chill marking the onset of the malady; the disease is generally made up of the progressive increase in the intensity of its phenomena—an imperceptible transition conducting us from its initial stage to its climax and decline. And, indeed, the termination is not an abrupt one—there is no rapid defervescence; it occurs slowly and progressively, unless in a few exceptional cases, where the temperature falls below the normal standard.[2] Lastly, relapses are oftener the rule than the exception in this disease.

We have yet to discuss a question that has been variously answered by different authors. Is the fever subordinate to the disease of the joint? Or is it, on the other hand, independent of any local lesion?

Certain it is that fever often persists after the arthropathy has entirely disappeared; but in such cases it is almost always maintained by some latent visceral disease, as, for example, an endocarditis or a pericarditis.

Again, fever may precede symptoms developed on the part of the joints; but here, also, it is often (we do not say always) induced by one of those visceral affections which sometimes set in before the articular manifestations of rheumatism.

Still, the febrile movement is often of great intensity when there is a small number of affected joints. Here is an unknown element that escapes us, seeming to justify the opinion of Graves, Todd, and Fuller, who regard fever in rheumatism as *primary*, and not *secondary*.

We have now regarded rheumatic fever from a general standpoint. Let us consider it, then, in detail, along with the accessory phenomena of the febrile state.

The *pulse*, whose frequence, according to Louis,[3] does not exceed ninety or one hundred pulsations per minute, presents special characters not met with, in other febrile diseases, to the same extent. The artery is of large volume, as in certain cases of anæmia, according to Monneret, Todd, and Fuller.[4] The sphygmographic tracings express this wonderfully well; they show an enormous amplitude,[5] and quite a pronounced dicrotism of the pulse—indeed, a remarkable resemblance to the pulse of aortic insufficiency. It is understood, of course, that, when cardiac disease supervenes, these characteristics undergo profound change.

The *secretions* in acute rheumatism are deserving of special attention. *Perspiration* stands in the first rank here, and is remarkable, in general, for its abundance and extreme acidity, especially about the affected joints.[6] The sudamina contain a fluid whose reaction is manifestly acid, and this property of rheumatic perspiration resists alkaline treatment—even in very large doses—during quite a long time. To these acid exhalations must undoubtedly be ascribed the sourish smell given off from rheumatic patients. But we cannot affirm with precision what principle, from a chemical point of view, determines this reaction; it has been attributed to lactic

[1] Wunderlich: Pathol. und Therapie. Vol. iv., p. 621.
[2] Hardy: Thèses de Paris. 1859.
[3] Louis: Recherches Anatomiques, Pathologiques et Thérapeutiques sur la Fièvre Typhoïde. Vol. i., p. 443. 1841.
[4] Monneret: Loc. cit., p. 53.
[5] Marey: Physiologie Médicale de la Circulation du Sang, p. 545.
[6] Williams: Principles of Medicine. Third edition, p. 194. London, 1856.

acid, but the proof of this is insufficient. Simon states that, in the course of acute articular rheumatism, there is acetic acid in the sudatory secretion; and this, according to the same writer, does not occur in the normal state of affairs. But Schottin has shown that, even in a condition of health, sweat not only contains acetic acid, but also butyric acid and formic acid.[1]

Saliva, according to Fuller,[2] becomes acid in acute rheumatism; and besides, all the fluids of the economy are, according to this author, remarkable on account of their distinctly acid reaction; the serous effusions into the pericardium and into the joints are also of an acid reaction. In a few cases I have remarked this latter occurrence, but only in exceptional cases; and this also occurs in gout. Let me add that the intra-articular fluids, sometimes acid, are also, in some instances, alkaline.

The condition of the urine is particularly deserving of attention, since here we find a distinctive difference between rheumatism and gout. Upon immediate inspection, the urine is scanty and very highly colored; upon cooling, it throws down a copious deposit of urates of a brick-red color.

Upon analysis, there is a marked diminution in the watery part (explained by the abundant perspiration), and an increase in the solid ingredients. Urea and coloring matter are especially found in large proportion; the occurrence of the latter probably corresponds to a destruction of the blood-globules greater than that in any other phlegmasia.

The percentage of uric acid is increased, it being found in the proportion of 0.85 to the litre, according to Parkes,[3] and 0.75 according to Garrod.[4] Here is offered another point of contrast with gout.

There is a diminution in the proportion of chlorides, not so great, however, as in pneumonia; and, lastly, there is well-marked acidity, though nothing proves that this arises from the presence of an excess of lactic acid.

A final characteristic of the general bodily condition in acute articular rheumatism is the intense anæmia developed in a few days after the commencement of the disease, even in those cases where no recourse has been had to the antiphlogistic method of treatment. There is certainly a similar result in phlegmasiæ (on account of the destruction of the blood-corpuscles), but to a very much less extent. Todd, O'Ferral and Fuller in England, Canstatt in Germany, and Monneret and Piorry in France, have dwelt with great insistence upon this point. In acute gout the condition of things is quite opposed to these phenomena, and we cannot help seeing therein a further difference between that malady and the one under present discussion.

II.—Comparison between Acute Articular Rheumatism, Gout and Subacute Rheumatism.

Following this summary description of acute articular rheumatism, we are prepared to point out the differences and the analogies which it presents, either with gout or with subacute rheumatism.

First.—Concerning *acute gout*, there is only one analogy to be pointed out here; this is the irregular and paroxysmal progress of the disease. Everywhere else we discover nothing but differences.

Thus, in gout, the temperature is lower (so far as can possibly be af-

[1] Donders: Physiol. Vol. i., p. 450. [2] Fuller: Op. cit., p. 517.
[3] Parkes: On Urine, p. 286. London, 1860.
[4] 13.12 gr. in 2.1135 pints (Parkes); and 11.58 gr. in 2.1135 pints (Garrod).—L. H. H.

firmed in the present state of our knowledge), and fever is not so intense; it seems to be more rigorously subordinate to the number of joints affected, and only assumes a truly high degree in general gout.

The pulse no longer presents the special characteristic that we found to prevail in rheumatism.

Perspiration is much less copious, and does not possess the acidity we have mentioned.

The urine differs from that described in rheumatism, although it is the same in appearance; but when we make an analysis of it, far from being any excess, we rather find, in gouty subjects, a diminution in the quantity of uric acid.

Finally, anæmia is never marked from the onset in cases of acute gout.

Second.—Again, *subacute* articular rheumatism occurs with the characters of the acute form; but the febrile movement is less intense, as are all the accompanying phenomena; the anæmia, however, being quite as marked as in acute articular rheumatism. On the other hand, the articular affections are more permanent, contrasting thus with the extreme unsettledness or mobility of the local manifestations of acute articular rheumatism. Lastly, we may draw a distinction between these two forms of rheumatism in respect to their duration; this is from six weeks to two months, according to MacLeod, in the subacute variety; while in acute rheumatism, the duration has been variously estimated, although the figures are always much smaller than those we have just enumerated; indeed, Professor Bouillard puts it at from eight to fifteen days. Legroux at one-and-twenty; Chomel and Requin at twenty-eight; and, according to Lebert, it varies in length with the method of treatment, and may even extend over twenty-eight or thirty-five days.

III.—Pathological Conditions of the Blood in Acute and Subacute Articular Rheumatism.

The composition of the blood in articular rheumatism differs considerably from what exists in cases of acute gout, and this is unquestionably one of the most important distinctions which differentiate these two diseases.

The clot of blood drawn from one who has acute articular rheumatism is, you know, resistant and retracted, resembling the buffy coat of pleurisy, or, as Sydenham somewhat familiarly puts it, *they are alike as two eggs.*

The investigations of Nasse, Simon, Andral and Gavarret, Becquerel and Rodier, furnish the explanation of this occurrence; they found a considerable increase in the proportion of fibrin which might reach seven or eight parts in one thousand, instead of three, the normal standard. At the same time there is a considerable diminution of the red blood-corpuscles. In this respect the composition of the blood in acute articular rheumatism may be regarded as the type of inflammatory blood, and differs essentially in its composition from that of the blood of gouty subjects.

Finally, let me add, that in those suffering from rheumatism the serum of the blood is alkaline, the proportion of urea is normal, and no excess in uric acid is found. This fact, which to-day is beyond all dispute, possesses a degree of importance impossible to be exaggerated.

But does not the blood in acute articular rheumatism present a few special characters belonging to it exclusively? Does it not contain some pathological product, some material that is foreign to the normal constitution, explaining why acute articular rheumatism differs in so many ways

from ordinary phlegmasiæ, which are nevertheless accompanied by the same kind of changes in the composition of the blood?

Many analogies are adduced in favor of such an hypothesis; but as yet they do not rest upon any positive fact.

In the last century, Van Swieten, Baynard,[1] and many other physicians made rheumatism dependent upon a particular acrimony of the blood, a retention of acids and salts that should have been eliminated through the kidneys. An hypothesis more in keeping with the data of modern chemistry has recently been advanced in England. It is supposed that lactic acid—the normal product of the disassimilation of fibrous tissue—is formed in excess and gives rise to all the phenomena we have just described. This opinion is upheld by Prout, Williams, Todd, and Fuller; but it does not stand on a firm basis of fact. Yet Richardson, after having injected lactic acid into the veins of dogs, found articular lesions and disease of the heart in the animals. His experiments, however, were repeated in Germany, and it was stated that cardiac disease in the canine species was of very great frequence, apart from any artificial interference. In this way it seems well established that the endocardial lesions existed previously; and as for the articular lesions, you must remember that they are affected, in a large number of cases, by various kinds of poisoning.

One circumstance deserves to have attention called to it in closing this rapid sketch. *Inopexia*—excessive coagulability of fibrin, independent of all excess in its amount—is present in the highest degree in acute articular rheumatism; hence the exceeding frequence of vascular thromboses and fibrinous cardiac vegetations in the disease.

On the other hand, in many grave cases we notice an entirely opposite condition. We then find at the *post-mortem* the blood to be fluid and black;[2] it no longer reddens from contact with the air, and it is here especially that the fluid exudations into serous cavities have a markedly acid reaction, as we have several times attested.

Finally, let me recall to you that, in many individuals, rheumatism seems to be allied with the hemorrhagic diathesis.

To summarize, we think we have shown that articular rheumatism, in all the various forms it may assume, constitutes one and the same morbid species, a species essentially distinct from gout. Acute, chronic, and subacute rheumatism, the latter serving as a transition between the two extremes, are actually but one and the same disease. We have endeavored to prove this by a study of the articular lesions and general characteristics of the disease; we shall find a new proof in the study of the *visceral lesions*, which will form the subject of our next lecture.

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[1] Baynard: Philosophical Trans. (abridged). Vol. iii., p. 265.

[2] Vogel: Virchow's Handbuch der sp. Path. und Therapie. Vol. i., p. 479.

LECTURE XIV.

VISCERAL AFFECTIONS IN ACUTE AND CHRONIC ARTICULAR RHEUMATISM.

Summary.—Comparison of the Visceral Diseases of Gout, and those of Acute or Chronic Rheumatism—Tardy Development of Visceral Affections in Gout; their Early Development in Acute Rheumatism—These Lesions manifested still later on in Chronic Rheumatism—Difference in the Nature of Visceral Lesions in Rheumatism and Gout—Cardiac Disease in Rheumatism—Rheumatic Pericarditis—Rheumatic Endocarditis—Modifications in the History of this Disease, Caused by the Progress of Modern Histology—Structure of the Internal Membrane of the Heart—Inflammatory Lesions of Endocarditis—Principally Located upon the Valves—Description of the Pathological Process—Tumefaction of the Endocardium; Vascularization of this Membrane—Result of this Pathological Condition—Capillary Embolism—Lesions of Canalization—Typhoid State—Chronic Stage of the Disease—Multiple Affections the Consequences of these Lesions—Ischæmia, Localized Gangrene—Ecchymotic Spots—Cerebral Softening—Fibrinous Deposits in the Spleen, Liver, and Kidney—Various Complications of Acute Articular Rheumatism—Cardiac Lesions may likewise occur in Subacute and Chronic Rheumatism—Lesions of the Respiratory System—Pleurisy, Pneumonia, and Pulmonary Congestion—Asthma and Emphysema—Pulmonary Phthisis—Lesions of the Urinary Apparatus—Nephritis—Albuminuria—Cystitis—Lesions of the Nervous System—Cerebral Diseases—Medullary Affections—Abarticular Lesions of Various Kinds—Muscular Pains—Neuralgiæ—Derangements of the Visual Apparatus—Cutaneous Diseases, Eczema, Psoriasis, Prurigo, Lichen, etc.

GENTLEMEN:—To-day we shall consider the abarticular affections of rheumatism. We shall at first endeavor to compare them with the changes which gout induces on the part of our internal organs; and then we shall ask ourselves whether the visceral lesions of acute rheumatism are met with, possessing the same characteristics, in the chronic forms of the disease.

During the course of an attack of acute gout, derangements of a purely functional nature are, for a long while, the sole indications that the viscera are suffering; they leave no material impress behind them, and it is only when incessant return of the attacks begins to clothe the malady with a chronic form, that we see those permanent changes develop which daily become graver and graver.

In articular rheumatism it may be said that things follow a nearly inverse order. Indeed, one of the prime characteristics of this disease (at least in the acute form) is an unusually early development of certain visceral lesions (endocarditis, pericarditis, etc.), which frequently manifest themselves at the first attacks—indeed, scarcely waiting until the disease has passed through its initial stage before they are quite distinctly marked. There is no longer, in these cases, any question of purely functional derangements, but of permanent lesions modifying the texture of organs, and almost always leaving behind them indelible traces of their occurrence.

Primary chronic rheumatism differs from the acute form in this regard. The existence of visceral disease here is so unusual that several authors have called it in question, and it may be said that the more the disease

tends to assume the chronic form, the *rarer* it is to see similar lesions developed during its progress.

Another fact to be emphasized here is that the visceral affections of rheumatism are only analogous to those of gout in a gross aspect; in reality, the lesions are essentially different.

Thus, the cardiac derangements—at first purely functional—that may supervene in cases of gout, are localized in the muscular fibre, when they become transformed into permanent lesions; we then find a condition of fatty degeneration.

In acute articular rheumatism, on the contrary, the cardiac diseases are inflammatory lesions expending their potency upon the endocardium and pericardium, only attacking the muscular tissue of the organ secondarily. The organic changes they so often leave behind them are the result of this inflammatory action. It is well known that, in nearly one-half the cases where there are permanent lesions of the auriculo-ventricular valves, acute articular rheumatism is the acknowledged cause.

Later on in the course of these lectures we shall dwell more particularly upon the *cardiopathies*, which are, to a certain extent, an integral part of acute articular rheumatism. They may be considered as one of the most characteristic features of this disease; the frequence in such cases being, in fact, so great, that when acute endocarditis or pericarditis is seen coexisting with an otherwise ill-defined articular affection, we are in the great majority of cases justified in ascribing the train of occurrences to rheumatism.

We shall, therefore, chiefly concentrate our attention upon those cardiac lesions whose recognition is of such great importance; the other visceral diseases of rheumatism are of much less interest from the special standpoint we have assumed; nevertheless, we shall say a few words concerning them.

I.—Endocarditis and Pericarditis in Acute Articular Rheumatism.

Rheumatic pericarditis is a disease which has been known for a long time; it can certainly be asserted that, to-day, all the points in its history are familiar to us. We know that it is an inflammation of the serous membrane enveloping the heart, and that this inflammation generally results in the formation of a sero-fibrinous exudation which, in a few exceptional cases, may assume a hemorrhagic or purulent character.

Farther on we shall point out the symptoms which, collectively, reveal the existence of this condition, and the particular circumstances in which it develops.

The history of endocarditis has, on the other hand, been singularly changed by the progress of histological study, and rheumatic endocarditis has particularly taken part in the transformation.

At an epoch not yet very remote, the endocardium was represented as a serous membrane, and the lesions it presented were thought to possess the characteristics of an inflammation of a serous membrane. In his *polypoid carditis*, it is well known that Kreisig described the secretion of plastic lymph as one of the principal characteristics of the disease; and it is also known that in the remarkable study upon endocarditis, by Professor Bouillard, this author dwelt with emphasis upon the intense redness presented by the internal membrane of the heart.

At present we are aware that, in certain respects, these views are lack-

ing in exactitude. The endocardium is not identical in structure with serous membranes, and does not inflame in the same manner. But it was an exaggeration in the reverse direction when men went so far as to deny the existence of the endocardium. To simple fibrinous deposits some have ascribed all the lesions which occur in this disease (Simon). This is an ultra-development of an idea which Laennec had previously advanced.

There is a certain amount of truth in both these opinions. There is endocarditis, and the internal membrane of the heart can become inflamed; but there is no plastic exudation. On the other hand, the formation of clots in the interior of the cavities of the heart plays an important part in the disease; but this occurrence is always secondary, and ought never to usurp the first place.

Let us first examine the histological structure of the internal cardiac membrane: we shall then be better prepared to understand the changes of which it may be the seat.

The endocardium is essentially composed of a very thin layer of connective tissue, containing a certain number of elastic fibres, and covered with pavement-epithelium. According to Luschka, the endocardium is continuous with all the tunics of the vessels, but the majority of writers state that it is only continuous with their internal membrane (*tunica intima*).

The endocardium has no vessels of its own; but upon the cardiac parietes the subjacent capillaries are very close, on account of its great thinness. It is wholly different in the neighborhood of the valves; here the investing membrane is thicker—a few vessels, according to Luschka, being found ramifying between the two lips of the mitral valve; but these never exist in the sigmoid valves in their normal state.

Now, it is upon the valves—that is to say, upon the very thickest portions of the endocardium, upon the portions farthest removed from the blood-vessels—that inflammatory lesions are preferably located; they commence, too, upon the external surface.

In what, then, does the pathological process consist?

In the acute stage the morbid action commences by a tumefaction of the affected portion; small papillary elevations are formed, made up of pre-existing elements which have sensibly increased in size, and also of cells of new-formation along with embryo-plastic cells. The whole papillary elevation is filled with a fluid whose reaction is similar to that of mucus. This is the first period of the disease.

FIG. 19.—Acute endocarditis: section of the mitral valve. *a*, superior layer of the endocardium; *a'*, inferior layer; *b*, middle layer, whose vessels show a marked degree of hyperæmia; *c*, efflorescence (granulation) of the superior layer of the endocardium; *d*, fibrinous deposit ($^1/_{10}$). (Taken from Rindfleisch: Lehrb. der pathol. Gewebelehre, p. 186. Leipzig, 1866.)

In the second stage the elevations sometimes acquire a permanent organization; and again their extremities are ulcerated, this lesion being the consequence of a granular degeneration that must not be confounded with fatty metamorphosis. These little ulcers have truncated apices.

Later on, the reddened and swollen point becomes covered with a layer of fibrin more or less thick, according to the case. And, as you know, inopexia (a tendency to coagulation of the blood) is an habitual result of rheumatism, as well as of the puerperal state and certain peculiar cachexiæ.

Valvular vegetations of the endocardium are the result, then, of inflammation of the tissue itself and of a secondary deposit of a fibrinous layer.

But, while this action is progressing, the vessels are developing. In the mitral valve, where they already exist, they become more apparent; and in

the sigmoid or semilunar valves they are of completely new formation, or at least neighboring capillaries send out prolongations into the parts destitute of vessels, as occurs in the cornea when that becomes the seat of inflammation ; and in this way arborescent vascularity may be found appearing about lesions which have invaded the cardiac orifices.[1]

It is very important to study the consequences of this pathological condition. First, however, let us see what are its immediate results.

In some instances the changes go no farther than this ; and then there are no lesions of canalization. Sometimes the fibrinous deposit softens, breaks down, and the detritus formed gives rise to capillary embolism. This is an essentially clinical side of the question and a wholly new aspect of it, whatever may be said to the contrary.

Again, and finally, the ulceration deepens, and then we have valvular perforations which give rise to the most diverse lesions of canalization ; the union of several of these openings may lead to the detachment of a fragment of the valve, and thus arises an embolism of greater or less size. Do not forget that valvular aneurisms are sometimes produced, located either at the mitral orifice or at the sigmoid valves.

In some cases the process undergoes a modification from causes unknown to us. Then pus may be formed, but this is a rare occurrence ; we oftener witness the development of deleterious material which comes from other parts and infects the mass of blood, giving rise to typhoid symptoms. In the majority of such cases we say that it is an instance of ulcerative endocarditis ; but, properly speaking, the ulcerative form of endocarditis is not necessarily accompanied by septicæmia.

The ulterior consequences of endocarditis are met with in the chronic stage of the disease. The phlegmasic activity spreads and changes its nature ; the whole valve becomes indurated, giving rise to a shrinking and toughening of it ; hence, insufficiency follows. At other times adhesions form between the diseased valve and the margins of the orifice, sometimes inducing an insufficiency, sometimes, on the contrary, a stenosis.

There are, occasionally, compensatory lesions, as has been satisfactorily proved by Jacks ; contraction of one of the semilunar valves, for example, left a void which, in some cases, was filled in by an elongation of the other two ; and in this way the mechanical portion of the lesion may be cured. I have, myself, met with unquestionable cases of this sort in the cadaver.

These are the elementary lesions of endocarditis. We shall not dwell in this place upon the appearance presented to the naked eye ; it is well known that these lesions occupy preferably the left heart and its auriculo-ventricular orifice, and that upon the mitral valve they especially invade the auricular surface and the parts which are in contact. At the semilunar valves these pathological depositions readily assume the form of a wreath of vegetations. What is of the greatest importance, however, to adduce here, is that there may often be no lesions of canalization. Frequently there is nothing produced except simple stigmata, which give rise to no appreciable functional derangement during life, and are only discoverable at the autopsy.[2] This occurs more frequently than is generally supposed ; and, while taking into account these rudimentary lesions, the proportion of coincidences between diseases of the heart and rheumatism is made too

[1] Ball : Du Rheumatisme Viscéral. Thèse de Concours pour l'Agrégation, p. 28. 1866.

[2] Charcot : Comptes Rendus de la Société de Biologie. Third series. Vol. iii., p. 269, 1862.

great. It is in the chronic form especially that it is important to note this point, as we shall presently see.

But, first, I wish to say a few words concerning the multiple affections arising from endocarditis, for this is not only one of the most original sides of the question, but one of the newest conquests of science.

At the present time we know that movable bodies may be detached from the diseased orifices, either at the expense of the fibrinous deposits, or the valves themselves, and, once thrown into the torrent of the circulation, they produce various symptoms in remote parts.

We must here distinguish between the results produced by displacements of large concretions and those arising from the transportation of almost molecular fragments.

First.—Arterial emboli, properly so called, may obstruct the circulation in vessels of the first order; the femoral and even the external iliac have both become suddenly impervious to the circulatory current on account of the presence of a large-sized clot from the heart.

When the arteries in the limbs are thus plugged, the result is generally an ischæmia terminating usually in gangrene. Watson, Tufnell, and several other authors, have reported instances of this in cases of rheumatic endocarditis.[1]

Second.—Capillary emboli—of infinitely greater frequence—may occur in nearly all the organs, and give rise to lesions of the most different kinds.

A.—When the *cutaneous* capillaries are obstructed, more or less extensive ecchymotic spots are produced.

B.—When the *encephalic* vessels are the seat of embolism, the result is softening, sometimes red, sometimes white, which is one of the most frequent causes of hemiplegia in those who have not yet attained an advanced age. When the obstructed artery has a large calibre, instantaneous hemiplegia is sometimes the consequence, and secondary softening almost always occurs. Kirkes has reported an extremely remarkable case of this kind.

Analogous symptoms are sometimes developed, when the principal arteries of the brain are found perfectly permeable after death. We observe, first, the appearance of all the symptoms of softening which occur in their usual order of progression; but after death we can find no lesion in the vascular canals. This anomaly may be explained in two ways. Actual obstruction has occurred in vessels of a large calibre; but the clot being reabsorbed, the artery has again become permeable, while the softening consequent upon the plugging has perished. On the other hand, it may be admitted that, very small vessels being thus obstructed, a cerebral disease has developed without the existence of any obstacle in the great encephalic circulation.

I have myself seen a case of this kind. The patient having first been in Trousseau's service, this eminent clinician admitted the existence of a cerebral embolism, following a valvular lesion of the heart. In this woman, who died later, in my ward, I found at the autopsy that there existed an old endocarditis with vegetations upon the mitral valve; but that no obstruction in the arteries of the base of the brain was found.[2] Cases of this kind may be explained upon the ground of reabsorption of the thrombus;

[1] Watson: Principles and Practice of Physic. Fourth edition. Vol. ii., p. 314. Tufnell: Dublin Quarterly Journal. Vol. xv., p. 371. Goodfellow: Transactions of the Medico-Chirurg. Society of London. Second series. Vol. xxviii. 1862.

[2] Trousseau: Clinique Médicale de l'Hôtel-Dieu. Vol. ii., p. 587. Bouchard: Comptes Rendus de la Société de Biologie, p. 111. 1864.

this reabsorption is sometimes complete at the time of making the autopsy; but in other cases slight vestiges of the obstructing clot are still found, which thus enable us to discover its nature to a certain extent.

This form of lesion is so frequent after rheumatic cardiopathies, that Lancereaux in his thesis attributes more than half the pathological cases he collected to this cause.[1]

C.—The *spleen* very frequently becomes the seat of capillary emboli, giving rise to infarctions having a cone-like form, the apex pointing toward the hilum, after the well-known manner of distribution of the vessels in this organ.

We ourselves had an opportunity to observe an interesting case, wherein this lesion manifested itself in consequence of a rheumatic affection of the heart. The patient was a man, twenty-four years of age, who was first attacked with acute articular rheumatism and then with the chronic form. During life he had a double "rasping" murmur at the apex. He frequently complained of intense pain in the region of the spleen, which organ had acquired considerable size. He died with the ordinary symptoms of heart disease.

At the autopsy fibrinous vegetations were found upon the mitral and semilunar valves, and two fibrinous deposits in the spleen, one having a considerable size, the other a little smaller.

D.—The *kidneys* may also undergo analogous changes: Rayer, not knowing their origin, however, has nevertheless given an excellent description of them under the name of *rheumatic nephritis*. (Figures 2, 5, 6, and 7, of *Plate F* of the atlas of the "Traité des Maladies des Reins"—A Treatise on Diseases of the Kidneys—are examples of *rheumatic nephritis* in patients suffering from heart disease.)

Capillary embolism of the kidney gives rise to changes which have been accurately described in the thesis of Dr. Herrmann.[2] They consist chiefly in a fatty degeneration which occurs around the injured point, and which is, later on, followed by a flattened cicatrix.

E.—The *liver* itself has no immunity from these occurrences, but this organ is affected only in rare instances.

F.—To these lesions arising from arterial or capillary emboli, we must add the septicæmic phenomena which sometimes develop from rheumatic cardiopathies. We then see the symptoms of the *typhoid state* supervene, the *deep jaundice*, and the *symptomatic intermittent fever* which is associated with acute articular rheumatism.[3]

II.—Endocarditis and Pericarditis in Subacute and Chronic Articular Rheumatism.

Gentlemen, now that we have thus traced the history of endocarditis and pericarditis in acute rheumatism, we shall endeavor to show you that these complications do not exclusively belong to this form of the disease.

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[1] De la Thrombose et de l'Emboli Cérébrales: Thèses de Paris. 1862.

[2] Des Lésions Viscérales Suites d'Embolie: Thèse de Strasbourg. 1854.

[3] When the right heart is the starting-point for these migratory clots, we may have pulmonary emboli, sometimes causing almost instant death. A case of this kind is reported by Goddard-Rogers. It was a case of acute articular rheumatism with endocarditis, in which the patient was suddenly seized, on the fifteenth day, with an attack of orthopnœa, inducing death in the period of ten minutes. At the *post-mortem* the pulmonary artery was found plugged with a large clot that had come from the right heart.—The Lancet, p. 19. London, 1865.

First, they are quite frequently met with in subacute rheumatism, in spite of the celebrated law of Professor Bouillard, at once so rigorous and so tyrannical a law, if thus I may be allowed to express myself.

Endocarditis and pericarditis are undoubtedly more frequent in acute and poly-articular rheumatism. Valleix, Latham, Bamberger, and Fuller are in full accord, in this respect, with the French clinical *savant*.

Nevertheless, cardiac complications are met with quite often in cases where a few joints only are affected, or where the febrile movement is not at all intense. West found this the case in children ; and Walshe, Ormerod, and Garrod affirm it for adults. If I may be allowed to offer my own experience in this connection, I have seen several cases of subacute rheumatism, where an autopsy has been made, coinciding with disease of the heart. One of these cases is recorded in Dr. Ball's "Thèse d'Agrégation."[1]

But the question is to prove that these lesions may exist in chronic, in nodular rheumatism, properly so called.

First.—It must be remembered that rheumatic *endocarditis* is very often a latent disease during life, but it almost always leaves behind it traces that can be recognized after death. Hence, one must not always expect to meet with palpable murmurs in patients suffering from chronic rheumatism, but pathological anatomy enables us to establish, in this respect, a bond of relationship between the acute and the chronic form of articular rheumatism, proving once more that there is but one and the same disease, notwithstanding the diversity of its pathological manifestations.

Cardiac lesions are quite frequently found in nodular rheumatism. A case of this kind has been described by Romberg in 1846.[2] Two other cases are found in the thesis of Dr. Trastour and in mine, in a collection of forty-one observations. Since that time, attention having been drawn to the circumstance, cases have multiplied. A few years ago, Beau, in a clinical lecture demonstrated the coincidence of aortic stenosis in a young girl with nodular arthritis ;[3] and Dr. Ollivier, when in Professor Grisolle's service, observed a case where a man, twenty-three years old, presented the characteristic deformities of nodular rheumatism, and gave evidence of a change in the aortic semilunar valves.

It is very often the case that these patients have had a previous attack of acute articular rheumatism ; but I have gathered quite a large number of observations in which endocarditis has developed in patients with chronic rheumatism when the disease has never taken the acute form. Two of these cases are found in the thesis of Dr. Ball.[4]

The first case was that of a *concierge*,[5] a woman sixty years of age, whose right hand bore the characteristic deformities of chronic rheumatism. She ascribed this disease to the dampness of the lodge which she had occupied so long.

She came to the infirmary of the hospital for the first time on account of a right hemiplegia, and gave no evidences then of any cardiac implication ; after two months' treatment she went back to her dormitory.

But the second time she came, there were evident signs of heart-trouble. The symptoms of the disease became rapidly worse, and death soon occurred.

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[1] Du Rheumatisme Viscéral, p. 64.
[2] Klinische Ergebnisse. Berlin, 1846. A similar case was described by Todd, in 1843.
[3] Gazette des Hôpitaux. July 19, 1864. [4] Op. cit., p. 121 and following.
[5] A *concierge* occupies a loge—a sort of box—at the door of buildings where dwell many families, and is door-keeper and janitor combined.—L. H. H.

At the *post-mortem* examination there was found *a general adhesion of the pericardium to the heart, and this lesion was evidently of recent origin*, for the pericardium could easily be removed from the subjacent tissue. The heart had attained an enormous size, and *wreath-like vegetations* were seen *upon the semilunar aortic, and the mitral valves, whose ventricular surfaces presented a remarkable degree of vascularity.*

In the second case the patient, eighty-four years old, died in the infirmary of the Salpêtrière, from cancer of the liver and stomach. In this woman was found a dry arthritis of the shoulders, elbows, and knees. The heart was large, flaccid, and loaded with fat; and *upon the aortic valves were palpable traces of an old endocarditis.*

It thus seems evident to me, in view of the facts I have just presented, that we may meet with organic lesions of the heart in the course of primary chronic rheumatism.

Second.—It is probable that *pericarditis* is frequent in chronic rheumatism, for of nine autopsies I made in 1863, with Dr. Cornil,[1] I encountered it in four. Indeed, we now have a case of this kind under observation, and Dr. Mauriac has reported a very remarkable one of the same sort at the Ménages Hospital.

Let me give you an analysis of this interesting case.

A woman, seventy-one years of age, who has long suffered from chronic articular rheumatism and pulmonary catarrh (bronchitis), is suddenly seized with intense dyspnœa and a distinct pain in the right side. Percussion elicits a slight increase in the area of precordial dulness, and on auscultation we hear a *rude, rasping pericardial friction-sound* over the whole lower half of the sternum, where it masks the normal heart-sounds. Under the influence of appropriate treatment this patient recovered in the space of six weeks, after having experienced inflammatory symptoms in those articulations that were already diseased.

This case may be compared to one observed by Martel, in Dr. Barthez's service at the Sainte-Eugénie Hospital. A child ten years old, suffering from chronic articular rheumatism, developed a pericarditis marked by friction-sounds in the precordial region. This condition, however, did not last very long. The rheumatism underwent an exacerbation during the progress of the pericarditis; and subsequently this little patient presented, in the highest degree, all the characteristic deformities of nodular rheumatism.

Since the period when my attention was directed to this question, I have frequently seen cases of pericarditis coincident with the exacerbations of arthropathies in those attacked with chronic rheumatism. Thus, the instances I have just recorded are in perfect accord with my own personal observations.

To sum up, then, endocarditis and pericarditis unquestionably occur in some cases of chronic articular rheumatism; and these conditions there exhibit the same characteristics as when present in acute rheumatism. They occur preferably during the period of exacerbation of the disease, and when it somewhat approximates, as it were, the acute form. Finally, these two diseases are, in general, of a less serious nature when they develop during a chronic articular rheumatism.

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[1] Cornil: Mémoire sur les Coincidences Pathologiques du Rheumatisme Articulaire Chronique. Mémoires de la Société de Biologie. Fourth series, vol. iv. 1865.

III.—Some other Abarticular Diseases in Chronic Rheumatism.

Cardiac affections are not the only visceral manifestations of the rheumatic diathesis. Let me remark, however, that it is important to observe that the other usual complications of acute articular rheumatism are more rarely met with in the chronic variety than endocarditis and pericarditis. But, on the other hand, we shall soon learn that certain affections accompany the chronic form that are almost wholly unknown to the acute, as one would naturally surmise.

In this connection we shall rapidly review the principal systems of the economy.

The Respiratory Apparatus.—*Pleurisy* occurs in subacute and chronic articular rheumatism, but it is much rarer than in the acute form.

Acute pneumonia is a frequent complication of acute arthro-rheumatism; I have never met with it in the chronic form. I have certainly seen some cases of chronic pneumonia in rheumatic patients, but the latter were cachectic.

Rheumatic pulmonary congestion, that dire condition rendering the patient liable to sudden death, may be encountered in subacute or chronic rheumatism. Dr. Ball records a very remarkable case of this kind in his "Thèse d'Agrégation."[1]

Certain thoracic affections occur especially in chronic and ill-defined forms of articular rheumatism. *Asthma*, followed by *emphysema*, is as common in this disease as in gout. I have seen this complication twice in the Salpêtrière.

The first case was that of a woman, sixty years old, who for twenty-five years had been a washerwoman.

Her asthma dated back ten years. At first the attacks were few and nocturnal. At the time of recording the case the paroxysms were so closely recurrent, so commingled, that the patient passed the greater part of both day and night sitting on the bed, breathing with intense difficulty and distress.

There were always sibilant râles that could be heard at a distance, and auscultation showed great feebleness of the vesicular murmur over the entire surface of both lungs. The expectoration was scanty.

This woman never had been attacked with acute general articular rheumatism; but several times, during a period of seventeen years, she had arthritic pains accompanied by swelling of the joints. The parts principally affected were the knees, shoulders, and the metacarpo-phalangeal joint of the index finger of the right hand. There were no characteristic deformities in this case, but a well-marked "crackling" noise was observed upon motion of the knees.[2]

The second case was that of a woman, sixty-six years of age, who after having lived for a long time in a very damp lodging, seemed to have had an attack of acute articular rheumatism. She remained in bed for six weeks, and two months after she got up she could not walk without crutches. Since that time there were occasional attacks of lumbago and flying articular pains. At the age of forty there was a new attack of subacute articular rheumatism. A last attack of the same kind occurred four years before she came into the service.

The menopause occurred in her fifty-sixth year, and it was a short time after this that oppression and a sense of constriction appeared. At the

[1] Op. cit., p. 61.

[2] Op. cit., p. 129.

commencement of the pulmonary disease, she was for nearly two months unable to lie upon her bed. The attacks, always of long duration, were at first considerably remote from one another; then they drew nearer and nearer together, so that, for about three years, the oppression and constriction became permanent, and were much increased by walking.

When the thoracic disease assumed such a degree of intensity, the articular pains became vaguer and more flying. There were no deformities of the joints; but very distinct "crackling" in the knees.

After quite a long stay in the infirmary, this woman died, an extensive œdema of the lower limbs meanwhile having been developed.

The autopsy revealed:

First.—Hypertrophy of the *right* ventricle, without any valvular lesions, and without a trace of old or recent pericarditis.

Second.—Articular lesions, almost identically the same as those of acute articular rheumatism, but without synovial effusion into the joint, and a much stronger-marked velvety condition of the osseous surfaces.

Third.—Well-marked redness and evident vascularity of the mucous membrane of the bronchi as the pulmonary lesions; these tubes contained a considerable quantity of greenish and tenacious mucus.

This woman's son has long been asthmatic.[1]

It is clear that in this interesting case there was first an acute articular rheumatism, and then a subacute, which finally ended in the chronic form. The coincidence of asthma with articular manifestations in this patient is of capital importance from the standpoint whence we are now considering the subject.

The existence of a *laryngitis* allied to acute rheumatism has, you know, been pointed out long ago. Garrod has described in addition a particular form of chronic laryngitis occurring in nodular rheumatism.

Has *pulmonary phthisis* any relation to chronic rheumatism? This is still a mooted question, and one that we purpose to discuss farther on in the course.

Urinary Apparatus.—In acute rheumatism there is a special *nephritis*, which we have just described. Kidney-changes are almost the rule in gout. In chronic rheumatism *albuminous nephritis* is quite frequent in the advanced stages, according to the investigations which, together with Cornil, I made concerning this matter. These lesions, besides, are always met with in those who are markedly cachectic.

Vesical implication is quite rare in acute articular rheumatism. Vesical gout, and those burning pains experienced by rheumatic subjects during micturition, must not be confounded, the last-named sensations arising from extreme concentration of the urine.

In chronic rheumatism, on the contrary, *cystitis* is quite frequent, especially in those who have long been confined to their beds.

The Nervous System.

A.—*Cerebral diseases.*—We have described the striking analogies existing between *rheumatic encephalopathy* and the cerebral symptoms accompanying gout. *Rheumatic insanity*, described by Burrows, Mesnet, and Griesinger, is quite often associated with the subacute forms of rheumatism.

Symptoms of this kind, in chronic rheumatism, have been described by Fuller and Vidal. But these cases are quite rare.

[1] This case is recorded, but incompletely, in Dr. Ball's thesis.

Megrim, so common in the intervals between the attacks of gout, is also present in the course of chronic rheumatism.[1]

B.—*Medullary affections.—Chorea*, whose relationship to acute articular rheumatism is so well established, does not seem to be present along with the chronic form; at least I have never seen it occur in the latter disease.

Several diseases, however, that arise in the spinal marrow, are met with in the chronic variety of rheumatism.

Paralysis agitans, or, at any rate, the *tremor*, sometimes accompanies partial or general chronic rheumatism.

I have seen several cases of *locomotor ataxy* coincide with dry arthritis or nodosities of joints. But, since we now know that the arthropathies of ataxics may manifest themselves at the beginning of the disease, it becomes extremely difficult to estimate the exact part played by the rheumatic diathesis.[2]

As for *paraplegia*, it is rarely present in either acute or subacute rheumatism, and we must be well on our guard against confounding *articular pains* of spinal diseases with rheumatism in the joints; for lesions of the spinal cord occasionally induce painful swellings of the articulations, as Mitchell, Morehouse, Remak, and several other observers attest.

Certain lesions yet remain to be described, which, though not located in the internal viscera, are still abarticular.

We have seen that gout sometimes causes affections apart from the joints, besides its visceral diseases; and it is the same in the acute and chronic forms of rheumatism.

Thus, the *muscular pains* of gout, followed by cramps and retraction of the muscles (Guilbert), may likewise be met with in acute rheumatism; but they are much more frequent in chronic rheumatism.

Sciatic and *trifacial neuralgiæ*, although occurring equally in gout and rheumatism, yet especially belong to the subacute and less intense forms of both diseases. They may also be met with in Heberden's rheumatism; Dr. Bastian communicated a case of this kind to me.

The *visual* mechanism may be affected in rheumatism as well as in gout. We have already spoken of gouty *iritis* and sclerotitis. Eye-affections are rare in acute rheumatism; but it is quite the contrary in subacute (Garrod, Fuller) and chronic rheumatism (Cornil). There is generally an *iritis*, but obstinate cases of *conjunctivitis* may also occur. A palpable alternation between the ocular phenomena and the symptoms referable to the joints is frequently seen.

Finally, *skin diseases*, so well known in gout and acute rheumatism, are also met with in chronic rheumatism. They are not very common in the *intenser* forms of this disease, being principally encountered in partial chronic rheumatism. Bazin observed, also, that cutaneous diseases are the more tenacious and obstinate the less marked are the arthropathies.

We have very frequently found the skin diseases described by Bazin in chronically rheumatic patients: *eczema*, *numular psoriasis*, *lichen*, and *arthritic prurigo*.

Indeed, we have often seen *erysipelas* allied to nodular rheumatism.

In our next lecture we shall take up the symptomatology of chronic rheumatism.

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[1] On this subject consult Dr. Malherbe's thesis, p. 45. Paris, 1866.

[2] Charcot: Archives de Physiologie, p. 162. 1868.

LECTURE XV.

SYMPTOMATOLOGY OF CHRONIC PROGRESSIVE ARTICULAR RHEUMATISM.

Summary.—Three Fundamental Types of Chronic Articular Rheumatism—In Reality Constituting but One and the Same Disease—Chronic Progressive Articular Rheumatism, or Nodular Rheumatism—Frequently Confounded with Gout, from which it Essentially Differs—Is Preferably Located in the Smaller Joints.

Arthropathies arising from Nodular Rheumatism—Often Resemble, at the Commencement, those of Acute Rheumatism—Spasmodic Retraction of Muscles—Altered Positions, *Attitudes Vicieuses*—Permanent Disorders—Pain—Crackling—Bony Deformities—Joints which are Preferably Affected—The Hands almost always the First Attacked—Symmetrical Invasion—Mode of Succession of Cases of Arthritis—In Young Subjects, frequently Generalized from the Commencement—In Older Patients has a Progressive Course—Consecutive Deformities of the Limbs—Two Principal Types, their Varieties.

Progress of the Disease—Secondary Changes—Atrophic Form—Œdematous Form—Loss of Movement.

Deformity of the Lower Limbs—Of the Vertebral Column—Deviation of the Head—General Invasion of all the Joints.

Mode of Production of these Lesions—Various Opinions—Spasmodic Contractions—Accessory Causes.

General Symptoms—Hæmatology—General Reaction—Rapid Development—Slow Development.

Gentlemen:—Until now we have regarded chronic articular rheumatism in only one of its aspects. The pathological anatomy of this disease has been, until the present moment, the object of our study; but it is time to enter upon the clinical view of the question, and to present you with the symptomatic evidences that reveal the existence of this affection.

We have already dwelt upon the necessity of recognizing at the outset three fundamental types in respect to anatomy; and it seems equally necessary to establish this distinction in regard to symptomatology. The division is, as you already know, 1st, nodular rheumatism, or progressive chronic articular rheumatism; 2d, partial chronic articular rheumatism; and 3d, Heberden's rheumatism.

These are not three distinct diseases, but three particular forms of one and the same affection. It is none the less indispensable to separate them, for the nature and chain of symptoms, the prognosis, and even the treatment, varies in each of the three types of chronic rheumatism.

We shall devote this lecture to *progressive chronic articular rheumatism.*

From a medical point of view, nodular rheumatism is the most interesting of the three types we have enumerated. Often mistaken for gout, it has sometimes received the name of *gouty rheumatism.* We have already demonstrated to you that a radical distinction must be made in this respect. Rheumatism ought never to be confounded with gout; but all the varieties of articular rheumatism are part of one and the same pathological species, and should be assigned to the same category.

Nodular rheumatism is to the other forms of chronic rheumatism what acute general rheumatism is to the subacute or partial variety. It especially affects the smaller joints, those of the hand in particular. It is a disease that too often defies all the resources of our skill, and gives rise to deplorable infirmities.

We shall first describe the *arthropathies* induced by this disease; and secondly, the symptoms arising from the constitutional condition.

1. *Arthropathies.*—In the first stage the articular phenomena—if we except their locality—differ in nowise from those in acute or subacute rheumatism. The affected joints are the seat of pain, redness, heat, and swelling—the same characteristics as in acute rheumatism; but here the symptoms are much less intense, and are infinitely more stable. Let us also observe that there is no œdema, no desquamation, as we have in gout.

This train of symptoms, however, is frequently joined by a new phenomenon, even at the very commencement—a phenomenon which is far from appearing with the same degree of intensity in the acute form: we mean that spasmodic contraction of the muscles which produces such strange and sometimes permanent distortions of the diseased members.

In the second stage we have the development of lasting disorders. Tumefaction has occurred in the soft parts, productive sometimes of an hydrarthrosis, sometimes of a thickening of the synovial membrane and subserous tissue; it has likewise occurred in the hard parts, and then we observe the development of osteoids or foreign bodies, common enough in some joints, though rare in others, the osseous swellings deforming the heads of the bones, and finally, subluxations of the articular extremities.

The pain, either spontaneous or induced, now becomes permanent; it may be located in the articulation itself, the body of the bones, or the adjacent muscles, in the form of agonizing cramps.

Then it is that we hear the crackling or crepitation due to the eburnation of the bony surfaces, if the joint still preserve a certain degree of mobility; although very often the rigidity arising from fibrous ankylosis and retraction of fibrous-tissue becomes perceptible likewise at this epoch. Lastly appear those bony deformities that follow certain laws and rules, which we shall consider farther on in our course.

And now let us see what joints are preferably invaded. This disease, as we have already said, has a predilection for the smaller joints. The larger articulations are attacked only in advanced stages of the disease, although even in cases of a general implication, the shoulder and hip are frequently spared. You must bear in mind, however, that the upper extremities are almost always the first attacked; this is just the reverse of gout, in which the upper limbs are only secondarily affected as a rule.

Nodular rheumatism's mode of invasion also presents distinctive peculiarities. *Symmetry* is here the rule, as Budd observes, a statement confirmed by Romberg, and also verified as to its accuracy by myself. Gout, as you know, is far from following any such course as this. Let me remark, however, that in a few exceptional cases chronic rheumatism is asymmetrical, and the rarity of this occurrence induces me to call attention to it. In this instance one side of the body is first attacked, and subsequently the articular lesions become generalized.

A distinction no less marked exists between gout and nodular rheumatism in regard to the parts that are first attacked. In gout it is the great toe; in nodular rheumatism the metacarpo-phalangeal articulations of the index and middle fingers are the first invaded, and this, too, on both sides

of the body simultaneously, in conformity to the law of symmetry we have enunciated. Do not forget, however, that this disease begins in a large joint much more often than gout, and this fact alone frequently aids us in making a diagnosis. In connection with this question let me adduce also a few statistics:

First.—Hands and feet alone, smaller joints.....	25 times.
First.— — — — — The great toe.....	4 times.
Second.—Hands and feet along with a large articulation..	7 times.
Third.—A large joint at first, and later on the fingers....	9 times.

Let us now look at the mode of succession of arthritises in nodular rheumatism. In the majority of cases they develop from the periphery toward the centre; the fingers are first implicated, then the elbow, then the shoulder. This regular succession, however, is met with only in those cases where the disease develops slowly. In young patients, from sixteen to twenty years of age, this affection, on the contrary, is often general from the outset; but in older subjects, from forty to sixty, it follows the progressive course we have just described. In the first case the disease is evolved rapidly; in the second it develops slowly. But nothing is absolute in this respect, since often, at the menopause, the symptoms occur abruptly and the malady presents all the traits of an acute disease.

We have yet to describe the deformity of the limbs that results from this morbid action. They are especially marked in young subjects when spasmodic pains are present and muscular retractions are excessive. We do not speak now of deformities of the joints themselves, but of the faulty positions arising from a change in the relation between the various segments of the limb.

These deformities are almost always identical; they obey regular laws. In the case of the upper extremity they may be divided into two principal types, with secondary derivatives.

In both cases the hand is more or less exaggeratedly pronated. This characteristic belongs to each in common; but they differ in many other respects. We shall give a succinct description of all of them.

First type.—This is the one most frequently met with.

It is characterized:

a.—By flexion at an obtuse, right, or even acute angle of the terminal phalanx upon the middle phalanx.

b.—By extension of the middle phalanx upon the proximal phalanx.

c.—By flexion of the proximal phalanx upon the metacarpal heads.

d.—By flexion, at a less obtuse angle of the metacarpus and carpus upon the bones of the forearm.

e.—In a great number of cases by an inclination, *en masse*, of all the phalanges toward the cubital (ulnar) side of the hand, and then a twisting in the opposite direction of the middle upon the proximal phalanges. The former of those two lesions is often one of the first deformities to signal the advent of this disease.

This type presents two varieties. In the first, most of the characteristics we have described are preserved; but the proximal and middle phalanges are in the same plane, have the same axis, and form a single column.

In the second, flexion of the terminal upon the middle phalanges is absent, and then the fingers seem hollowed or excavated, starting from the projecting metacarpal heads.

Second type.—This is marked:
a.—By extension of the terminal phalanx upon the middle phalanx.

FIG. 20.—First type.

b.—By flexion of the middle phalanx upon the proximal phalanx.
c.—By extension of the proximal phalanges upon the metacarpal heads.

FIG. 21.—First variety of Type 1.

d.—By a more or less pronounced flexion of the carpus upon the bones of the forearm.

FIG. 22.—Second variety of Type 1.

e.—In certain cases, by a deviation, *en masse*, of the proximal phalanges, which are visibly inclined toward the ulnar side of the hand.

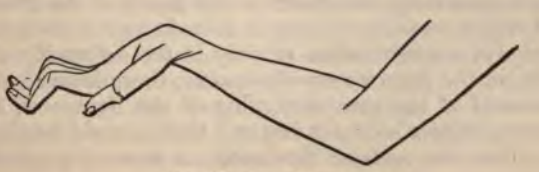

FIG. 23.—Second type.

This type, like the preceding, may present two varieties.

In the first, there is flexion of all the articulations of the hand, constituting, after a fashion, a kind of scroll.

FIG. 24.—First variety of Type 2.

FIG. 25.—Second variety of Type 2.

In the second we observe the same thing, except that, in addition, there is extension of the middle upon the proximal phalanges.

Hitherto we have only considered the deviation of the fingers of the hand; what becomes of the thumb?

Here, as elsewhere, the metacarpo-phalangeal articulation is especially altered. The first phalanx of the thumb is often flexed, though sometimes extended.

The other articulations of the upper extremity partake, in a certain measure, in these deformities.

Thus, flexion of the elbow is more or less marked—indeed, is sometimes exaggerated; extension is impossible.

The forearm is pronated.

There is more or less complete flexion of the carpus and the metacarpus upon the forearm, with projection of the ulna and radius.

Finally, the shoulder is sometimes rigid, and the whole upper extremity is fixed against the thoracic wall.

We shall next consider in what manner, and in what order, the articulations of the upper extremity are invaded.

Two cases may present themselves.

A.—In young subjects the disease progresses rapidly, and spasmodic contractions of the muscles being very marked, the deformities are distinctly exhibited. There is projection of the heads of the bones following upon subluxations that have occurred in the joint.

Thus, in the *second* type, there is considerable projection of the bones of the forearm behind the carpal bones; there is a subluxation of the heads of the proximal phalanges, in front and to the outside, producing quite prominent projections upon the back of the hand; and there is a slightly marked subluxation of the middle phalanges forward upon the proximal phalanges, the heads of which bones bulge upon the palm of the hand. Finally, on account of prolonged flexion of the terminal upon the second phalanges, the small condyles of the latter project behind. Attention must be called to the fact that the osseous vegetations around the digital articulations do not constantly circumscribe the heads of the bones: sometimes there are tubercles, sometimes more or less elongated needles. At any rate, these osteophytes contribute but slightly to the deformity of the joints.

And lastly, in the form we are discussing, there is more complete immobility, on account of the greater rigidity of the ligaments and fibrous tissues: fibrous ankylosis soon results, and thus a rapid wasting of the parts is induced. The term *nodular rheumatism* is scarcely applicable to cases of this sort.

B.—In older subjects the disease develops more slowly, and the power of movement is partly retained. Deviations from the normal are much less strongly marked, and the size of the heads of the bones and of the osseous stalactites is greater.

There is, in reality, a sort of inverse proportion between the degree of deviation and the size of the bony swellings, as well as the extent of the deformities resulting from the latter.

When the disease is prolonged beyond a certain period of time, changes are produced which are the results of this state of affairs. Atrophy of bone, atrophy of muscles and wasting of the soft parts are the commonest occurrences in such cases.

We can distinguish, however, two opposite forms here: in the *atrophic* variety (Vidal) there is induration of the skin—a kind of scleroderma: the cutaneous investment is cold, pale, smooth, and shining, no longer wrinkling.

In the other form an œdematous infiltration, simulating elephantiasis, occurs in the member, and this swelling is frequently accompanied by inflammatory symptoms; these phenomena are especially manifested in the lower limbs. In all cases the muscles of the limbs finally undergo a certain degree of atrophy and fatty degeneration on account of the immobility of the parts.

The immediate result of these changes is that the patients are debarred of movement in the upper extremities, and are no longer able to feed themselves. Then appear those ingenious inventions—those long forks whose form and dimensions vary according to the infirmity of the sufferer, but whose object is always to enable them to convey food to the mouth, thanks to the more or less restricted power of movement they still possess.

In this connection let me remark that the right hand, with which the patients continue to feed themselves, does not present in its deformities so regular a type as the deviations in the left, which, in the vast majority of cases, becomes absolutely immovable.

And now let us look at the articular lesions that may occur at other points in the skeleton.

The lower limbs are occasionally free from implication, and this when the hands have suffered the most marked changes. But such cases are rare, and generally the lower limbs are deformed symmetrically; there is deviation of the segments, and they become incapable of producing ordinary movements.

The hip-joint usually preserves its freedom, but the same cannot be said of the knee. In the majority of cases there are flexion of the thigh upon the abdomen, and flexion of the leg upon the thigh. The principal deformities are as follows:

The lower end of the femur projects in front of the head of the tibia.

The internal condyle of the femur becomes protuberant.

The patella, thrown to the outside, rests upon the external condyle.

The head of the fibula projects backward.

Complete ankylosis at the knee-joint is, however, a rare occurrence; but bony swellings are almost always developed there, and foreign bodies may be met with in great abundance in this locality.

In the tibio-tarsal articulation, on the other hand, ankylosis is very frequent. The foot may be abducted, then resting upon the external border,

thus simulating *talipes valgus;* and it may, on the other hand, be carried in abduction: then we have a *talipes equino-varus.*[1]

The great toe is carried outward so that it covers the other toes.

Important deformities may likewise occur on the part of the cervical vertebræ. In several cases that I observed myself in the Salpêtrière, I have seen the head bent forward and flexed upon the sternum, so that the chin nearly touched the chest. The movements of the head were very limited, and caused crepitating or crackling sounds when attempted. In the majority of these cases the neck was noticed to be enlarged posteriorly.

And it is in this way that most of the joints may be attacked in one and the same individual, in a case of general chronic rheumatism; and then the unfortunates are condemned to remain in bed during the remainder of their existence. They sometimes live more than twenty years in this miserable condition.

The *mode of production* of these deformities yet remains to be studied. Some physicians ascribe them to a providential action (Beau) which aims at palliating the intensity of the pain; others believe them to be the result of attitudes which the patients instinctively assume (Trastour).

I profess, myself, a diametrically opposite opinion on this subject. In the majority of cases these deformities come from *spasmodic,* and, as it were, *convulsive, muscular contractions.* They are produced by a sort of reflex action whose starting-point is in the diseased joints.

I shall not dwell long upon this subject. The arguments that I have previously advanced, reinforced by those of Crocq, appear to me to have incontrovertibly proved that these results are produced in precisely this manner.

In this regard we may adduce:

First.—The very form of the deviations or deformities; they are manifestly forced actions.

Second.—The resistance that the patients offer to these spasmodic retractions, which, although impotent to prevent them, is sufficient proof that they are involuntary.

Third.—The general aspect of the deformities, which certainly accords with idea of muscular spasm: they are, indeed, *deformities* or *deviations as a whole,* even when the joints brought into play are not all implicated.

Fourth.—Finally, the presence of the same deformities in cases where the joints are in nowise affected; they can then be ascribed to the spasmodic contraction of muscles alone.

Thus, in paralysis agitans, in congenital cerebral atrophy and in atrophy of the interosseous muscles, the parts occasionally assume a form in every respect the same as that observed in chronic articular rheumatism.

Moreover, it would be impossible to deny the existence of accessory incentives in addition to the fundamental cause of rheumatic deformities. The weight of the limbs, the greater or less laxity of the ligaments, have a correspondingly important part to play in the production of these deviations from the normal; but these various conditions are insufficient in themselves to induce similar effects without the concurrence of muscular retraction. Do we not see in certain cases of hydrarthrosis exceedingly great laxity of

[1] This is an exact and almost literal translation of the original paragraph. I think the word external should be internal (*interne* in place of *externe*), and that the last *ab*duction should read *ad*duction.—L. H. H. (See Erichsen's Surgery. Seventh edition. Vol. ii., pp. 369–371 inclusive.)

the ligaments of the knee, without any subsequent deformities resulting therefrom? On the contrary, the part remains absolutely flaccid.

As we have seen, the deformities that have just been described do not belong exclusively to rheumatism. But the changes in the joints, the crackling or crepitation, the hydrarthrosis, the fibrous ankylosis, and consequent rigidity of the articulation, the symmetry of the lesions—all these distinguish the effects of chronic rheumatism from those produced by paralysis agitans and various other diseases. But *gout* gives rise to the same spasmodic contractions, and to the same deformities and deviations. In his "Treatise on Gout," Guilbert has dwelt at length upon these muscular retractions.

The presence of tophi, however, which accompany in the majority of cases this kind of deformity in the gouty, is a truly pathognomonic characteristic. In rheumatic patients nodosities occasionally perforate the cutaneous investments; but the part exposed is bone, and not a chalk-stone, as in gout.

General Symptoms—Course of the Disease.

A.—Hæmatology and a chemical study of the secretions have as yet given only negative results.

Musgrave says the blood is buffed (*couenneux*); it certainly is in the acute forms, but we never find any uric acid in it. I have, myself, examined the blood in thirty-five cases of chronic articular rheumatism, without ever discovering the least trace of this substance.

In a case reported by Böcker, the chemical analysis furnishes most interesting results. This case is cited as one of gout, but it may certainly be attributed to nodular rheumatism, for the extremities of the bones were *swollen*—a thing that never happens in gout. The blood and the urine were examined, and in the urinary secretion there was a marked diminution in the normal proportion of phosphate of lime, while, on the contrary, the blood contained *four times more* phosphate of lime than normally present. It appears that the ordinary quantity of uric acid was not increased—at least, the author is silent upon this point.

B.—From a standpoint of general reaction and the progress of the disease, we have to recognize two essentially distinct forms.

In chronic articular rheumatism that develops *rapidly*, the disease is usually in young subjects from sixteen to thirty years of age, or in women who are pregnant or in the puerperal state; moral emotions and impressions of intense cold may likewise exert a certain amount of influence.

In such a case we see a large number of joints simultaneously attacked.

Muscular retractions are more pronounced, pains are very severe, redness and swelling are quite marked, and the disease is less unstable than in its opposite type.

The general symptoms are those of acute or subacute articular rheumatism; we notice marked elevation of the temperature, manifest acceleration of the pulse and profuse perspiration. It is in these cases especially that cardiac affections are met with.

It might be admitted here that rheumatism, at first acute, has later on assumed the chronic form; this opinion, which has been upheld by various writers, may be true in a few cases. Dr. Ball brought me a patient in whom the metacarpo-phalangeal articulations were attacked during the

course of an attack of acute articular rheumatism. The result was a deviation of the fingers toward the ulnar side of the hand; and this man, who to-day remains wholly recovered, still carries these characteristic deformities of nodular rheumatism.

We think, however, that, in the vast majority of cases, such examples are chronic rheumatism from the outset, but that the course of this chronic form presents some of the characters of acute rheumatism.

After a certain period of time, a remittent fever is developed, resembling hectic fever; and then we witness the occurrence of a series of exacerbations followed by long intervals of remission.

In cases of this kind the disease usually lasts but for a comparatively short time. After two, three, or four years the joints almost completely cease to be painful, and the case remains, then, in that condition. Sometimes, indeed, these deformities cease to exist after having been produced. We have observed cases of this kind ourselves; unfortunately, they are only too rare. In this way, also, a woman who was at first attacked with subacute febrile rheumatism affecting the shoulders and the metacarpo-phalangeal articulations, three months after the commencement of the disease suffered spasmodic retraction of the fingers; this secondary deformity lasted during an entire year, and then the patient recovered.

Chronic rheumatism that develops *slowly*, generally occurs in older subjects, from forty to sixty years of age: this disease is often met with at the time of the menopause. Geist [1] described this form under the name of senile gout. It is here especially that we meet Haygarth's nodosities of the joints.

One by one the articulations are invaded in succession; in these cases, therefore, the mode of invasion can best be studied. Pain is less intense and there is less redness; indeed, it is often wholly wanting. The deviations resulting from muscular contractions are less marked, but, on the other hand, the *deformities* of each joint are much more prominent.

Concerning the general bodily condition, it may be said that it is but rarely accompanied by febrile movement; only from time to time is a slight rise in the temperature observed. Finally, taken all in all, the prognosis is not so grave as in the preceding form.

We have compared these two extreme varieties in order to expose their analogies and their differences, but between these two forms there is a number of intermediate stages, establishing a gradual transition. Let me remark, too, that, notwithstanding the rule which we have enunciated, cases are met with in younger subjects that are the counterparts of those occurring in the aged: and the converse also holds good.

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[1] Klinik der Greisenkrankheiten. Erlangen, 1860.

LECTURE XVI.

SYMPTOMATOLOGY OF PARTIAL CHRONIC RHEUMATISM AND OF HEBERDEN'S NODOSITIES.

Summary.—Partial Chronic Rheumatism—Various Denominations it has Received—Does not Essentially Differ from Nodular Rheumatism—Its Particular Characteristics—Small Number of Joints Affected—Larger Articulations oftenest Involved—Insidious Advent—The Form Chronic from the Commencement—Articular Deformities—Diathetic Manifestations—Cutaneous Diseases—Visceral Affections.

Mode of Development—Occasionally succeeds Acute Rheumatism—May show Itself at the Beginning—Sometimes Generalized.

Articular Phenomena—Deformity—Pain—Absence of Sensibility on Palpation—Crackling or Crepitation.

Prognosis Comparatively not Serious—More or Less Complete Abolition of Movements—Spasmodic Retraction of the Muscles quite Rare—Extreme Rigidity of the Articulation.

Nodosities of Heberden—Independent of Gout—Are Located around the Articulation of the Terminal Phalanges—Lesions are Identical with those of a Dry Arthritis—Other Joints of the Hand frequently Involved, but to a Less Degree—This Affection Accompanies the Rheumatic Diathesis—It may, though very rarely, Coincide with Gout.

GENTLEMEN:—We shall complete the description of chronic rheumatism's symptoms by a study of the two chief forms of this disease following upon nodular rheumatism, in the classification we have adopted. We mean *partial chronic rheumatism* and *Heberden's nodosities.*

Partial chronic rheumatism, to which we shall first devote our attention, has received a large number of appellations varying with the different writers upon the subject. When located at the hip-joint, it is called *morbus coxæ senilis;* it has been denominated *senile arthritis,* and *dry arthritis,* when it had its seat elsewhere. It is the disease that produces the most characteristic deformities: it is *arthritis deformans.*

This disease does not essentially differ from the preceding; it is marked, however, by a few peculiar symptoms. Several of these we have already described. And now we wish to recall the most characteristic differences that separate these two forms of one and the same disease.

First.—A small number of the joints is involved, in comparison with what occurs in general rheumatism.

Second.—The larger joints, and even those which, in the preceding form, only suffer in very rare instances, are here the ones oftenest attacked.

Third.—The form that is chronic from the onset is here the rule. In the majority of cases it begins insidiously; pain is not very marked, and the affected members usually preserve their mobility—that is, to a certain extent. But, on the other hand, considerable deformity frequently occurs about the joints, on account of the exuberance of the osseous swelling; and besides, we sometimes meet with an hydrarthrosis sufficient to aid in the deformity. (*Hypertrophic form* of Adams.)

This disease has been especially studied from a standpoint of external pathology, and investigators have more especially devoted their attention to those cases where the disease was localized in a single articulation. Indeed, the diagnosis may then present the greatest difficulties; for this form of rheumatism may simulate a luxation, a fracture, and still more, a white swelling. These are questions that have always attracted the attention of surgeons; nevertheless, the study of this disease possesses a great interest for the physician—indeed, it involves the question of a disease almost always diathetic, which may frequently be accompanied by cutaneous affections,[1] and by visceral lesions (asthma and cardiopathies) whose signification is shown by the very fact of their coinciding with chronic arthropathies.

It may be observed, besides, that the localization of the disease in a single joint, the latent development of the malady and the extreme prevalence of bony swellings of new formation, are facts that are rather the exception than the rule, and facts that are especially striking from their very rarity. The arthropathies of partial chronic rheumatism very frequently appear with all these characteristics—in a very inferior degree, it is true, but nevertheless perfectly appreciable. This fact is of great clinical importance, for the well-established existence of these arthropathies may become an easily comprehended index of the rheumatic diathesis; and it is from this point of view that it is of significant interest to us.

I shall not undertake, gentlemen, to give you a description in due order and form of this variety of rheumatism; it belongs especially, as I have said, to the domain of surgery, and on that ground I do not wish to encroach. But I must needs acquaint you with certain of its characteristics, which, considered from the standpoint we have taken, possess the highest importance for us.

A.—Chronic partial rheumatism occasionally appears to succeed acute articular rheumatism, and thus becomes its consequence.

Adams, who has made a special study of this question, recognizes this mode of development of the morbid phenomena. In such circumstances it is one of the forms of nodo-articular rheumatism. Two varieties may occur: sometimes the rheumatism degenerates into a white swelling; sometimes it assumes the characters of arthritis deformans. The latter happens oftenest, and in this transition there is nothing to excite any surprise, since we know that acute rheumatism has, at its inception, the lesions of the chronic form.

B.—The commencement of arthritis deformans may be acute, while the rheumatism is partial from the onset. Adams[2] and Colombel[3] record examples of this, and I have myself observed a few such cases in the Salpêtrière. Thus, a woman fifty-three years of age had, at the beginning, acute partial arthritis, with fever and muscular retraction; then the disease persisted under the form of a dry arthritis; finally symmetrical pains occurred in both hands, and the disease passed into nodular rheumatism.

C.—On the other hand, the disease, as we have already seen, may commence slowly and insidiously, the symptoms remaining localized in a single joint from the start; but the real nature of the disease is in the end revealed by one or more arthritises, or of some abarticular manifestations allied to the rheumatic diathesis. There is now in the Salpêtrière a woman,

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[1] See Colombel: Recherches sur l'Arthrite Sèche. Thèses de Paris. 1862.
[2] Op. cit. Obs. xvii., pp. 44 and 301. [3] Op. cit. Obs. v., p. 71.

forty-six years old, who, after first having a dry arthritis of the hip-joint, was later on attacked by a similar condition of the knees. Another patient, a woman, sixty-three years old, first had dry arthritis of the right hip-joint, next of the left, and then a painful swelling of the left knee with crepitation; finally the nodosities of Heberden made their appearance.

D.—Lastly, quite a number of cases are met with where rheumatism, after having been stationary for a longer or shorter time upon a certain number of joints, becomes general. Two opposite transformations may be met with: at times partial rheumatism becomes general; and again, on the other hand, a single articular affection is observed to predominate in a case of general rheumatism, and the affected joint finally develops the lesions of arthritis deformans. Several examples of this are recorded by Adams.

Among others, he quotes the case of Dr. Percival, a very distinguished physician, who died in 1839, at the age of eighty-two, after having long suffered from this disease. In 1818 he had quite severe pains in the hands and wrists, accompanied by slight swelling. Two years later there was a little pain in the right coxo-femoral articulation, and gradually walking became extremely difficult to him. The limb was shortened and rotated externally; its movement was accompanied by well-marked crackling; and, when at rest, the limbs were crossed; he could not move from this attitude without inducing pain. Five years before he died analogous symptoms occurred in the left hip-joint.

Dr. Percival's death was caused by a disease of the bladder; and, in conformity with his last wishes, an autopsy was made by Dr. Colles, and the specimens were presented to the Pathological Society of Dublin. The articulations presented the most characteristic type of senile arthrocace;[1] the heads of the bones were flattened and eburnated, as were also both acetabula. The ligamentum teres had completely disappeared. The neck of the femur was shortened; the head of the left femur was reddened and vascularized, numerous bony deposits covering the capsular ligament.

In the case of a woman of sixty, who had suffered from nodular rheumatism for twenty-five years, the disease finally became localized in the knees, which were ankylosed at right angles, and presented all the typical symptoms of a dry arthritis.

But, in whatever way partial rheumatism originates, it is rather an infirmity than a disease when once it has attained the chronic stage. I make an exception here of the visceral lesions that may follow in its train. We must not forget, however, that acute exacerbations occasionally occur. The articular affection that has so long been painless, finally gives rise to intense sufferings, the skin reddens, and we mark an evident aggravation of the chronic symptoms.

The purely articular phenomena, when the disease is regularly established, are the following:

First.—There is more or less pronounced deformity of the joint; osseous ridges or crests, foreign bodies, and hydrarthrosis, are present.

Second.—The patient suffers spontaneous pains, quite vague in character, which pass off as he walks, but which finally become excruciating as the malady progresses.

Third.—No pain—or almost none—is produced by palpation or percussion—a peculiarity which distinguishes this arthropathy from those of a serious nature.

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[1] Arthrocace—ulcer of bone; (?) osteo-sarcoma.—L. H. H.

Fourth.—There is always more or less well-marked crackling or crepitation.

This disease, when exempt from all complication, in no wise threatens life; but it does away, more or less completely, with the movements of the parts affected. Nevertheless, the patients frequently continue to walk, though not without some difficulty. It is in these cases that the autopsy reveals striations upon the eburnated portions of the cuticular surfaces of the bones of the joint.

Spasmodic retraction of the muscles is very rare here, save at the commencement, but occasionally the ligaments are extremely relaxed; or, again, as we so often observe in the hip, there is very great rigidity of the articulation, on account of the deformity of the heads of the bones, or of their receptive cavities.

Nodosities of Heberden.—We now purpose to consider *Heberden's nodosities*, a special form of chronic rheumatism that has not yet been sufficiently described, and which, indeed, writers scarcely mention. Most physicians confound it with gout, without reserve or restriction.

We readily see that *nodular rheumatism* differs from true gout in many characteristics; but, in the case of Heberden's nodes, we are told that to doubt any longer would be unreasonable—the disease certainly is gout.

Personally, I hold the contrary opinion; and I designate this form of rheumatism by the name of *nodosities of Heberden*, because this author was the first to recognize that these lesions ought to be separated from those of gout.

"What," he says, in his "Commentaries," "what is the nature of the small, hard nodules, about the size of a pea, that we so frequently meet with in the fingers, especially upon their extremities, near the joint? *They have no connection with gout.*"

These little nodosities, as Heberden observes, have their seat at the articulation of the terminal phalanges. The digital extremity is in general but slightly deviated either to the right or left. There are two nodules on a level with the joint, which seems, besides, a little enlarged. There is rigidity, but no crackling or crepitation, in the diseased articulation.

In the majority of cases the advent of this condition is very obscure; but during attacks, or "fits," we have pain, heat, and temporary swelling of the soft parts. These are genuine attacks, which the patients often regard as gouty.

The minute anatomical lesions of this arthropathy have not as yet been described. According to the numerous investigations which I had the opportunity of making at the Salpêtrière, we find the changes of a dry arthritis here, as in the case of the two other forms of chronic articular rheumatism; I have ascertained this fact myself in many dissections. The articular cartilages undergo the "velvety" change; then they disappear, and we find in their place a layer of eburnated bony tissue. The articular surfaces are enlarged in all directions, on account of the formation of osteophytes which, while enlarging them, almost reproduce their normal form and contour. The pisiform tumors that, according to Heberden's description, are met with in the vicinity of the second phalangeal articulation, are nothing but osseous tubercles, which are normally present at the lower extremity of the dorsal aspect of the second phalanx; but there is a considerable increase in the volume of these tubercles from the apposition of new osseous layers. There is not a trace of urate of soda, either in the

deeper portions of the articular cartilages, or in the soft parts in the vicinity of the joints.

The remaining joints of the hand are usually affected, although to a much less extent. Contrarily to what occurs in nodular rheumatism, the lesions are best exhibited upon the articulations of the proximal with the second phalanges, and secondly, on the line of the metacarpo-phalangeal articulations.

These nodosities are interesting to us, since they reveal a constitutional condition, which is neither more nor less than *the rheumatic diathesis.*

This disease, of very frequent occurrence in the Salpêtrière, belongs especially to the senile period of life; but it must not be thought that it is never met with in young subjects; quite the contrary, we often observe it at far less advanced years, and this is an important point to demonstrate. Here is an hereditary disease; it may manifest itself in several members of the same family. It has palpable relations either with nodular, or especially with partial rheumatism; indeed, it is frequently seen to accompany arthritis of the hip or knee.

It sometimes happens that, in the same family, some have Heberden's rheumatism, others general chronic rheumatism, and still others partial rheumatism; another proof of the bond uniting these three forms of one and the same disease.

Heberden's rheumatism is often coincident with asthma, megrim, neuralgiæ—especially sciatic neuralgia, and muscular rheumatism. These manifestations may likewise alternate with acute attacks of this disease. It is not rare to meet with it in individuals suffering from cancer of the breast or of any other organ.

Lastly, these nodosities may occur in gouty subjects, as I had the opportunity of noticing quite recently. But in the case in question they preceded gout by several years.

LECTURE XVII.

ETIOLOGY OF ARTICULAR RHEUMATISM.

Summary.—The Principal Causes of Articular Rheumatism—Are Common to all the Forms of this Disease—Historical Pathology—Preponderance of Gout in the Writings of Physicians of Antiquity—Nodular Rheumatism, however, Already Recognized—Medical Geography—Acute Articular Rheumatism, a Disease belonging Especially to Temperate Climes—Unknown Around the Polar and Equatorial Regions—Chronic Articular Rheumatism Abounds in Temperate Climates, but likewise Occurs in Hot Countries—Heredity: its Incontestable Influence—Statistics taken from Various Authors—Age—The Classical Period for Acute Rheumatism between Fifteen and Thirty Years—Chronic Rheumatism Especially met with at Two Periods of Life: from Twenty to Thirty, and from Forty to Sixty—Sex—Men more Liable to Acute Articular Rheumatism—Women to Nodular Rheumatism.

External Causes—Wet Cold—Damp Habitations—Poverty, Insufficient Alimentation—Traumatic Causes—Blows, Falls, Phlegmon, Whitlow—Pathological Causes—Erysipelas—Angina—Scarlatina—Blennorrhagia (Gonorrhœa).

Uterine Functions—Chlorosis—Dysmenorrhœa—Menopause—Pregnancy—Prolonged Lactation.

Comparison between the Etiology of Rheumatism and that of Gout—These Two Diseases not Identical, but a certain Degree of Relationship Exists between Them.

Gentlemen:—The study of the causes which preside over the development of articular rheumatism furnishes new proof in support of the theory we have constantly upheld.

Indeed, upon the ground of etiology, we shall see the various forms of this disease approximate and become intermingled; we shall see them, on the other hand, diverge farther and farther from gout. And thus we shall establish the fact that the types apparently so different, which we have described heretofore, acknowledge essentially a common origin.

It must not be overlooked, however, that, according to a few observations, articular rheumatism has induced a predisposition to gout through the influence of heredity; and the converse of this seems, also, to be none the less certain.

I.—*Historical pathology and medical geography.*—While we were studying the history of gout, we stated that articular rheumatism had received but very little attention on the part of the physicians of antiquity, whose descriptions are almost wholly confined to gout; that they likewise confound the latter disease with rheumatism under the name of the *articular affection* (*articulorum passio*). Baillou is the first writer who gave a separate description of rheumatism; and the proof that this distinction required a long time to become established is to be found in the fact that it was not yet admitted in the first editions of Boerhaave.

The silence of the physicians of antiquity has caused many authors to imagine that rheumatism did not appear before modern times. But bones found in the ruins of Pompeii afford us more positive information on this

point than medical literature can furnish, at least so far as chronic rheumatism is concerned, since in many instances the characteristic lesions of this disease were actually found among the human remains. Most valuable and important information on this subject can be found in the "Osteologia Pompeiana," by Professor Delle-Chiaje of Naples. The plates accompanying the text banish every doubt from the mind of the reader.

A *medical geography* has yet to be written. Under the influence of certain preconceived ideas a deplorable confusion has arisen among all the diseases which trace their origin to cold; and it is easily understood how difficult becomes the task of criticism when observations made in remote regions are to be examined and tested.

It seems to be established, however, that acute articular rheumatism is a disease that abides more especially in temperate climates: it is unknown in the immediate vicinity of the poles and the equator. But this disease is quite frequently met with in hot countries: it often occurs in Egypt, according to Pruner-Bey, and in the East Indies, according to Webb; in the latter country it is frequently complicated by an endo-pericarditis.

At the Cape of Good Hope, the geranium's native home, the number of cases of rheumatism in one thousand sick in the English army, was *fifty-seven*; while in the rigorous climate of Nova Scotia there were only twenty suffering from rheumatism in the same number of patients.

Concerning chronic articular rheumatism, we possess no positive information, although it is at least certain that it prevails in temperate climes: in England, Ireland, France, Germany, and all Central Europe. But it may also occur in hot countries. In India, Malcolmson has found it among the Sepoys; and I myself have observed that it is a frequent disease in Naples.

II.—*Heredity.*—The study of this question possesses the greatest importance for the theory of rheumatism, since hereditary diseases are not accidental and transient,—they are in the very constitution of the patient; this is well seen in the case of gout.

The statistics of Chomel and Requin here establish the frequency of hereditary transmission; unfortunately, however, these authors confounded gout with rheumatism. But Fuller, who holds to the distinction between them, found the hereditary transmission of rheumatism 96 times in 300 cases, or a proportion of 29 per cent.;[1] while for gout the proportion is 50 per cent.

New investigations are indispensable in the case of chronic articular rheumatism. Still, all we do know of it tends to establish the fact that this disease often proceeds, because of an hereditary tendency, either from acute rheumatism, or directly from chronic rheumatism.

There can be no doubt upon this subject as regards *nodular rheumatism.* In forty-five cases of this type, Trastour states that the father and mother were rheumatic ten times, and three of the female patients had children who were already attacked with articular rheumatism. I observed an interesting case of this kind myself. There is now in the Salpêtrière a woman that has nodular rheumatism, whose *daughter* and *grand-daughter* are already suffering pains in the smaller joints. Here are three generations successively attacked with the same disease.

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[1] Prof. Charcot says: "96 fois sur 300, ce qui donne une proportion de 29 pour 100." It should read "87 times in 300 cases." See Reynolds: A System of Medicine. Vol. i., p. 938.—L. H. H.

In *Heberden's rheumatism*, heredity seems established according to personal observations of my own ; this is very frequently a family disease, a point to which Garrod has already drawn attention.

Concerning *partial chronic rheumatism*, the question is still under investigation, and I will not venture an opinion thereon.

III.—*Age.*—The classical time for *acute articular rheumatism* is between the ages of fifteen and thirty, although this disease is not rare in the earlier years of life; it develops in children of five and ten, and, according to West and the majority of writers, heart disease is more frequent in young people than in older subjects attacked with this form of rheumatism.

And here is a difference to be pointed out between this disease and gout, inasmuch as the latter is *rarely* developed before the twentieth year, as we have already stated (p. 87).

The acute form of articular rheumatism seldom occurs after the age of fifty. In one hundred and ninety-nine cases, Macleod saw it develop only once after fifty-five years of age ; and Fuller, out of two hundred and eighty-nine cases, observed only seven after fifty years of age.

I have seen two cases of this kind occurring after seventy years. The first case was a slight acute rheumatism, and the second was subacute febrile rheumatism of a very obstinate nature.

For *nodular rheumatism*, Trastour and myself have proved that there are two periods of life when one is more particularly liable to suffer an attack; it is from twenty to thirty, the epoch of complete development, and from forty to sixty, the time of the menopause, that this disease preferably appears. Thus Haygarth was wrong in ascribing chronic rheumatism almost exclusively to the menopause.

Nevertheless, this disease may manifest itself either before or after the periods we have named. We have many observations that are sufficient proof on this point.

Thus, Laborde brought before the *Société de Biologie* a little boy, eight years old, who had all the characteristic deformities of nodular rheumatism; the disease commenced when he was four years of age. I have already described, when speaking of rheumatic pericarditis (p. 130), the remarkable case that Martel recorded in the service of Dr. Barthez. I have myself observed the following cases :

A patient in the Salpêtrière, who had been reared in a damp dwelling, and who was attacked with nodular rheumatism at the age of ten.

Another in the same hospital, who had lived since her infancy in a damp lodge, was attacked when sixteen years old.

Finally, a man brought up in one of those deserted quarries on the banks of the Loire, that so often serve as dwellings, was attacked with nodular rheumatism at the age of twenty,

Partial rheumatism is—especially *arthritis deformans*—chiefly met with in individuals more advanced in life ; they frequently occur in middle-aged people. Only in the young are these diseases exceptional. Yet cases have been seen under thirty years of age.

As for *Heberden's nodosities*, we have seen that they may occur in young subjects, notwithstanding they especially belong to the senile period.

IV.—*Sex.*—Men are oftener attacked with *acute articular rheumatism*, than women. But there is no well-marked difference in this respect.

Nodular rheumatism is incomparably more frequent in females (Trastour,

Vidal); a convincing proof of this is afforded by a comparison between the inmates of Bicêtre [1] and the women in the wards of the Salpêtrière.

Partial rheumatism is perhaps more frequent in men; this is especially true for arthritis deformans.

Heberden's nodosities seem to be more frequent in the female sex; but this point is still an unsettled one.

External causes.—First: Wet cold.—A sudden and transient impression of cold can only be considered as an occasional, and in no wise as a specific cause of rheumatism. Eisenmann's mistake must be avoided: he wrote a work, "Erkältungskrankheiten" (diseases from cold), in which rheumatism comprises the whole pathology, or very nearly the whole.

It is nevertheless certain that, in predisposed individuals, cold has a powerful tendency to evolve either acute articular rheumatism, or chronic rheumatism of a rapid development.

But we are far from calling in question the influence of a *prolonged residence in a damp dwelling.* On the contrary, we here recognize the most efficacious cause of both acute articular, and of nodular rheumatism in particular.

About three-fourths of the women attacked with the latter disease ascribe it to the prolonged influence of damp cold. When stating this to be an invariable condition, Beau has undoubtedly exaggerated an unquestionable truth; but certainly this cause is present in the majority of cases. A dwelling on the ground floor, damp, dark rooms, wet garments, the paper peeling from the walls—this is the condition in which we find the homes of most of those who are attacked with chronic rheumatism; and besides, the patients have lived a long time in these sorry surroundings—for four, six, eight, and even *ten years.* In the vicinity of Chantilly there are genuine troglodytic dwellings; they are underground excavations in the interior of deserted quarries. Among the unfortunates who take refuge there, a large number, according to Beau, become subjects of nodular rheumatism. We need not wonder at this; but it must not be forgotten that, in many countries, people still live in subterranean abodes without appearing to experience any great inconvenience therefrom. This is the case, for example, in certain provinces in the Russian Empire, notably Georgia. During the "retreat of the ten thousand," you know that the Greeks, when they reached Armenia, found the inhabitants of that country lodged in excavations of this kind, and that in the middle of the coldest winter they were most comfortable in these well-warmed abodes.[2] Recollect, besides, that these are cold countries, and they only bury themselves underground to escape the severity of the climate. Now, we have seen that excessive cold is not at all favorable, in general, to the development of rheumatism. But, in temperate regions, it is more difficult to escape it when such conditions of life are present.

Yet the disease does not usually break forth without premonition; there is oftenest a period of incubation during which patients merely experience vague muscular pains. Articular manifestations frequently do not develop until three or four years after the cause has ceased to exist.

Second.—The influence of *poverty* and insufficient or improper food, upon the development of rheumatism, cannot be called in question: the in-

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[1] Bicêtre: asylum for old people and lunatics, in the neighborhood of Paris.—L. H. H.

[2] Xenophon: Ἀνάβασις. Book iv., § 48.

digent in the English and Irish workhouses offer numerous instances of nodular rheumatism, proving certainly that the disease is an especially plebeian malady, notwithstanding Haygarth's opinion to the contrary.

Third.—Traumatic causes, as in case of gout, may determine both the outbursts and the primary location of the disease.

We possess several observations where acute or chronic rheumatism has developed after a blow, fall, phlegmon, or whitlow, appearing first at the articulation nearest the injured part.

In a woman attacked with nodular rheumatism, the disease commenced in the right shoulder, which had previously received a severe contusion.

A butcher, already rheumatic, developed a phlegmon on the hand from a punctured wound of that member; an attack of acute rheumatism commenced in the wrist-joint, about the spot which was the principal seat of the phlegmonous inflammation.

A whitlow upon one of the fingers of a woman in the Salpêtrière marked the commencement of a nodular rheumatism in the joints nearest to the diseased part.

Partial rheumatism frequently develops after a fall or a blow; and it is here difficult to determine whether it is a general disease, or a purely local affection secondary to the external violence.

VI.—*Pathological causes.*—These often act in the same manner as external accidents. Acute articular rheumatism develops subsequently to a large number of different diseases.

In an individual who was already rheumatic, a facial erysipelas, contracted during an epidemic of this disease, was the starting-point for acute articular rheumatism. This case must be distinguished from those where erysipelas is a part of the manifestations of the rheumatic diathesis; for I could cite several instances of nodular rheumatism where the attacks of erysipelas have alternated with the articular symptoms.

A few facts tend to establish the existence of a rheumatic angina; but it is quite certain that a purely accidental angina has many a time been the starting-point of an attack of acute articular rheumatism.

The connection between articular affections and scarlatina is well known. Two kinds of cases must be distinguished in this respect. Sometimes scarlet fever induces the outburst of an articular disease in no way distinguishable from acute rheumatism; and again, on the other hand, it is an arthritis clearly of an opposite nature, benign in the vast majority of cases, according to a remark of Trousseau's, but which may become very grave and assume a purulent character, as Garrod has observed. The latter cases alone deserve the name of scarlatinal arthritis; the former belong to acute articular rheumatism, scarlet fever having here only played the part of a provocative. It is the same with blennorrhagia.

Is there a blennorrhagic (gonorrhœal) rheumatism? Here is a question that deserves our attention for a few moments.

In the majority of cases where articular symptoms are produced in consequence of an urethral disease, subacute arthropathies, to the number of two or three, are seen to appear in company with an iritis (Rollet); this is the classical type of blennorrhagic arthritis (gonorrhœal rheumatism).

But acute articular rheumatism with an endocarditis may also be observed to supervene upon a simple clap. A case of this kind was recorded by Brandes, and Professor Lorain communicated a second to me.

Finally, chronic rheumatism attended by deformity of the joints, may

also be occasioned by this disease (Garrod, Lorain, Broadhurst, Trousseau).[1]

How can we interpret these facts? Are the manifestations in these cases always rheumatic? Undoubtedly they are not. We know that the arthropathies which follow fevers are not always associated with this diathesis. The articular manifestations in glanders, small-pox and purulent infection (pyæmia) are clearly non-rheumatic in character. It may be, then, that there is a scarlatinal or blennorrhagic (gonorrhœal) arthritis independent of rheumatism. I am even convinced that this is frequently the case; but the articular disease that arises in such circumstances is often an *undoubted rheumatism*, developed secondarily to those affections which also, in certain instances, possess the inherent power of attacking the joints.[2]

VII.—*The uterine functions.*—All authors recognize the influence exerted upon the development of the various forms of rheumatism by the functions of the female genital apparatus. And this is true not only for the acute form, but also for the chronic variety of the disease.

Indeed, the appearance of the catamenia, the menopause, pregnancy, delivery, the puerperal state, and lactation, are causes which in women exert a powerful influence over the development of articular rheumatism.

And concerning this, let us enter somewhat into details.

Chlorosis is a base on which the articular manifestations of rheumatism are very apt to develop. Musgrave cites several cases of *arthritis ex chlorosi* that evidently belong to nodular rheumatism. The happy results arising from the administration of iron are affirmed in many cases of this kind.

It is well known that the *menopause* is frequently accompanied by a condition analogous to that of chlorosis in young girls, and it is likewise a settled fact that chronic rheumatism often develops at this period of life.

Pseudo-membranous *dysmenorrhœa* has been described by Todd among the diseases that accompany nodular rheumatism. It is perhaps not uninteresting to observe in this connection that dysmenorrhœa frequently accompanies the eruptions (*erythema nodosum*, for example) that are occasionally met with in acute and subacute rheumatism. In individuals already suffering from the malady, each menstruation has produced an exacerbation of the pain. The abrupt suppression of the menses, following upon some intense emotion, has occasionally been the starting-point for nodular rheumatism.

Pregnancy is likewise one of the causes of this disease. In a work which my colleague, Dr. Lorain, was kind enough to send me, are recorded several examples where acute, or subacute articular rheumatism, has appeared in women who were with child. In my *thesis* I have reported several cases of *nodular rheumatism* which developed under the same conditions; Todd had already noted the same coincidence.

It is not rare to see an isolated arthritis appear during pregnancy, and become general after delivery. Besides, acute rheumatism itself has been seen to occur during pregnancy (Chomel and Requin, Todd). It must not be forgotten, however, that multiple purulent arthropathies may be de-

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[1] Garrod on Gout, p. 545. Broadhurst in Reynolds' System of Medicine. Vol. i., p. 920. Trousseau: Clin. Méd. de l'Hôtel-Dieu. Vol. iii., p. 375. Professor Lorain's case was a verbal communication.

[2] See a discussion on this subject, occurring in the Medical Society of the Hospitals: Union Médicale, December 23, 1866, and March 5, 1867.

veloped in confinement. We think that these cases have been wrongly designated by the name puerperal rheumatism.

Another condition in which the various forms of rheumatism—subacute and chronic especially—may present themselves, is *lactation*, particularly when this is long-continued (Lorain, Garrod).[1]

It would be interesting to compare this etiology with that of gout, and exhibit the differences between the two; but time presses, and we must content ourselves with a general view of the question.

Generally speaking, it is true that the causes of gout are associated with comfort, excesses, and good cheer; in the same general way, rheumatism, especially the chronic form, has poverty, damp cold, insufficient food, and debilitating influences of various kinds, as its etiological factors.

But the contrast becomes yet more striking if we compare the diseases usually associated with gout, with those habitually accompanying rheumatism.

On the one hand we find diabetes,[2] obesity, and gravel,[3] whose relationship with gout we have already proved (pp. 75 to 79), and whose appearance in rheumatism is rarely observed; on the other hand, scrofula,[4] phthisis, and cancerous affections,[5] of frequent occurrence in chronic rheumatism, are very uncommon in gout.

Here, certainly, are differences between the two diatheses from an etiological standpoint; and yet, in spite of these great diversities which separate them, and which we have endeavored to make clear and prominent, they still offer remarkable analogies; indeed, they have frequently been confounded. And even when they have been differentiated, they must nevertheless be approximated in every good nosological classification.

It is certain, moreover, that a relation capable of being demonstrated in many ways, closely associates rheumatism and gout.

They sometimes appear in the same individual, who presents at the same time the lesions of both gout and chronic articular rheumatism.

Again, acute articular rheumatism occurs in a patient during his youth, and then gout is developed at the usual time therefor.[6]

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[1] On Gout, p. 568.

[2] In two hundred and twenty-five cases of diabetes, Griesinger, in his Studies of Diabetes, only *twice* found acute articular rheumatism. I do not think that diabetes has ever been observed as a complication of chronic rheumatism.

[3] I have seen uric acid gravel occurring in a woman with nodular rheumatism; but an examination of the blood at several different times, in this case, never revealed an excess of uric acid therein.

[4] Scrofula very frequently appears among the antecedents of individuals attacked with progressive chronic rheumatism. It is very common to see those patients with specific cicatrices of the neck. I could mention several cases where women who, during their youth, have had white swellings, and in whom, at a later period, nodular rheumatism has developed. In one hundred and nineteen cases of nodular rheumatism, Fuller (loc. cit., p. 334) states that twenty-three (or one-fifth of the whole) had mother, father, or collateral relations presenting evident signs of phthisis pulmonalis. This disease, I can avow, often carries off patients afflicted with nodular rheumatism; and in such cases it seems to me that the phthisis was remarkable for its slow development. In individuals attacked with acute rheumatism, phthisis is rare (Wunderlich, Hamernjk). Still, a coincidence of these two diseases is possible: Danjoy, who has directed attention to this point, thinks that the disease is then modified in its development—that it is retarded.

[5] I have often convinced myself, in the Salpêtrière, that the coincidence of Heberden's nodosities either with cancer of the breast or cancer of the uterus, is not an exceptional circumstance.

[6] See Baillou on this subject, vol. iv., p. 415. Was it this that made Juncker ask: *rheumatismus arthritidem ordine antecedit?*

Finally, a relationship may be established between them by means of heredity. Acute articular rheumatism occurs frequently in the children of the gouty (Heberden, Fuller, Todd). The children of the rheumatic often become subjects of the gout (Fuller). Indeed, heredity may show itself in the collateral branches: I have myself seen nodular rheumatism appear in a woman whose brother was gouty.

Do these relations, apparently so close, prove the identity of these two diseases? No, certainly not: at the utmost, let us admit the existence of a common foundation, a common basis, an *articular predisposition*, an *arthritic condition*, whence both affections take their origin.

LECTURE XVIII.

TREATMENT OF GOUT AND CHRONIC ARTICULAR RHEUMATISM.

Summary.—General Considerations Concerning the Treatment of Gout—Treatment of the Attacks or Paroxysms—The Expectant Plan—Quack Remedies—Colchicum—Advantages and Disadvantages of this Agent—Rules which should Govern its Employment—Narcotics: Hyoscyamus and Opium—Sulphate of Quinia—Iodide of Potassium—Tincture of Guaiacum—Topical Remedies—Leeches—Blisters—Moxa—Treatment of the Constitutional Condition—Alkalies—Their Various Properties—Sodium, Potassium, Lithium—Action of these Drugs—Cases where Alkalies are Contraindicated—Mineral Waters—Tonics and Stomachics—Treatment of the Local Affection: Chalk-Stones and Rigidity of Joints—Treatment of Abnormal Gout—Dietetic Regimen.

Treatment of Chronic Articular Rheumatism—Unsatisfactory State of our Knowledge upon this Subject—Treatment of the Acute Exacerbation—Opium, Sulphate of Quinia, Bloodletting—Alkalies—Tincture of Iodine—Arsenic Internally and Externally—Tincture of Guaiacum—Iodide of Potassium—Iron, Cod-Liver Oil—Blisters, Revulsives—Mineral Waters—Medical Art Powerless in the Majority of Cases.

GENTLEMEN:—We reach, to-day, the last part of our course. We have postponed the treatment of gout until the close of our lectures, so as to be able to compare it with that of chronic articular rheumatism. And this question we shall now discuss.

TREATMENT OF GOUT.

I.—*General considerations.*—Gout is a constitutional, hereditary disease, and primarily chronic, notwithstanding its acute manifestations.

But gout is also at times an acquired affection, now arising because of errors in diet, and now from other causes; this is a kind of spontaneous generation.

It may be concluded that hygienic influences are here placed in the first rank, and that therapeutical agents only occupy second place. Indeed, experience has long since demonstrated the truth of this statement.

We do not mean to say that the disease is radically incurable; examples of spontaneous cure are on record, but our art has not yet been able to reproduce with certainty the processes of nature.

The proper means do exist, however, for lessening the effects of the disease, and to avert its paroxysms; upon these, collectively, rests our treatment of the constitutional condition during the interval between the attacks or fits.

But we have a humbler, although more useful mission to fulfil. The periodical manifestations—the paroxysms—of both acute and chronic gout, are accompanied by terrible and often unbearable suffering.

Can we suppress these crises of pain, or at least diminish their intensity

and shorten their duration? This constitutes the treatment of a paroxysm of gout. Let us first consider the latter point.

II.—*Treatment of a paroxysm of acute or chronic gout.*—This is a question of treatment which is in great measure palliative in character. A few physicians have even gone so far as to proscribe all means of relief as dangerous and pernicious. This is Sydenham's school, which assumes the teleological standpoint; this great teacher says: "*Dolor acerrimum naturæ pharmacum.*" "Gout is gout's best remedy," said Mead; and Cullen prescribed "patience and flannel."

The partisans of the expectant plan base their belief on the inefficacy of known remedies, on the danger of their application, and especially on the relief experienced by the patient after the attack or fit. But to these arguments it may be answered that the inertness of capable men opens the way to quacks. The physician leaves a gouty patient—the quack seizes upon him. They appear with remedies which give almost instantaneous relief, and, if they sometimes induce grave symptoms, are yet often free from all real danger. Such are Reynold's elixir, Laville's anti-gout liquid, Auduran's wine, Lartigue's pills, etc., etc.

Now, I believe it is well established that all these so-called specifics owe most of their efficacy to the presence of *colchicum*. And thus it is the part of the physician to attentively study the therapeutic properties of this formidable agent, which sometimes affords the patient the greatest relief without any harm, and again induces serious symptoms that may end in death. No one, indeed—not even its bitterest enemy, denies its potency. It causes the gouty inflammation and the terrible pain that accompanies it to disappear as if by magic.

In this respect its action is nearly similar to that of quinine in intermittent fevers; and here, again, is one of the differential points between gout and articular rheumatism. In the acute form of the latter disease, Professor Monneret has already shown that colchicum is useless; and in the different forms of chronic rheumatism, I have assured myself that this drug possesses no advantages whatever.

How does it act in cases of gout? Since the sixth century of the Christian era, the ancients were acquainted with the advantages and disadvantages of colchicum. Alexander of Tralles tells us that, in his time, it was given only to those who were busy with their work and had no time to be sick. Demetrius Pepagomnenus, who lived about the year 1200, calls it *Theriaca articulorum.*

But the colchicum of the ancients is not our colchicum. They used the *hermodactyl*[1] (*colchicum variegatum*, Planchon); to-day we use the *colchicum autumnale.*

Fallen into neglect, this drug was again brought into notice and use by the effects of *Husson's remedy.* Everard Home extolled colchicum, which Störch had already brought into vogue for other diseases than gout. Later on its effects were carefully studied by Wandt, Halford, Watson, and Garrod.

All parts of the plant are used—bulb, seeds, and flowers. It is administered as the extract, wine, or tincture. The wine of colchicum-bulbs is administered in doses of from two to six grammes, twice or four times during the twenty-four hours (*thirty minims to a fluid drachm and a half*); the

[1] Hermodactyl: Mercury finger.—L. H. H.

acetic extract is prescribed in doses varying from five to fifteen centigrammes (gr. j.–ij., nearly).

A word concerning the physiological effects of this drug. In large doses it causes:

First.—More or less serious gastro-enteric phenomena.

Second.—A marked sedation or depression of the circulatory system, with a tendency toward algidity and slowing of the pulse.

Third.—Finally, nervous symptoms and a peculiar kind of drunkenness.

In small doses it merely creates slight nausea and a moderate retardation of the circulation.

Now, it is in small doses, at least when it is tolerated, that it acts favorably in gout; in its administration we must avoid the inflammatory phenomena on the part of the digestive tract; indeed, its action seems to be the more efficacious the less pronounced are its operative effects (*effets visibles*).

Its specific action is shown in the disappearance of the gouty inflammation and the pain that accompanies it; resolution occurs, as if by magic, at the end of eight to fourteen hours. It is far from possessing the same degree of influence over other inflammations and the various forms of articular rheumatism, as we have previously intimated.

What is its mode of action? This is a question which has not yet been solved. No effects have been attributed to the elimination of uric acid; this theory, upheld by Chélius, Maclagen, and Gregory, is combated by Garrod, Böcker, and Hammond. The latter base their opinion upon careful urinary analyses that seem to leave no opening for criticism.

Its sedative action upon the circulation has been adduced as a reason; but this is certainly not the secret, since it does not act in the same way in other inflammations.

Its purgative action is also out of the question, for its specific properties can manifest themselves without the occurrence of any intestinal evacuation.

We cannot, finally, ascribe it to its narcotic power, for even in this it presents an effect peculiar to gout.

Be this as it may, its efficacy is beyond all question. But we must look upon the dark side of the picture, and see what dangers the administration of colchicum involves.

It is beyond dispute that, when imprudently given, very serious results may be the consequence. What are the rules, then, that should govern its administration?

First.—Gout is a retrocedent disease, as we have previously proved. If, then, you abruptly suppress an attack or paroxysm, visceral derangements may be developed; but no such danger is to be dreaded when small doses are exhibited. Besides, colchicum ought not to be administered immediately upon the outbreak of the attack (Halford, Trousseau); several days should elapse before beginning its use. Finally, we should fear its irritating action upon the digestive tract; and this is another reason for not prescribing large doses.

Second.—Not only must we avoid the exhibition of large doses, but it is also necessary to suspend the drug for a time, since, in certain patients, its effects appear to be cumulative. Under such circumstances we may have reason to apprehend a sudden impression upon the nervous system; indeed, I am disposed to think that many cases where gout has apparently retroceded under the influence of colchicum, and induced death, are nothing but cases of colchicum-poisoning (Potton).

Third.—We must not accustom the patient to the use of this drug, for, in that case, he is obliged to take ever-increasing doses. There are colchicum-drinkers as well as opium-eaters and drunkards (Todd). Under these conditions a more or less profound change in the organism may be induced, and, on account of this influence, gout may pass into the *atonic state.*

Fourth.—Colchicum ought not to be administered in asthenic gout, but it may, nevertheless, be given with advantage in certain paroxysms of chronic gout. It is said to occasionally prolong the attack; but frequently, on the other hand, it seems to shorten the duration of the disease (Goupil, de Rennes).

Fifth.—The action of colchicum must be aided by a proper regimen (diet, rest in bed) and by adjuvants; salts of potassium and lithium should preferably be employed. Indeed, purgatives are often given with benefit; but the mercurials are contraindicated, experience proving that they have serious disadvantages.

There are cases in which colchicum may not be administered. But, despite this, the practitioner is not completely helpless. There are still other means which may be resorted to with success.

During an acute attack we may give, internally, the narcotics, especially hyoscyamus. Opium may also be prescribed, but it is disadvantageous on account of diminishing the secretions, and in this way hindering the regular development of the disease. It also induces, in certain individuals, effects which are out of all proportion to the dose exhibited. I have often seen these drugs (narcotics) cause distressing cerebral phenomena, and even induce uræmic symptoms in those who had pre-existing renal disease. We should be especially apprehensive of occurrences of this kind when gout has been of long standing, and the lesions of gouty kidney are already well-marked.

Todd records a very remarkable case of this kind.

The sulphate of quinine may likewise be administered with some chance of success; but its action in gout is far from being as efficacious as in acute rheumatism.

In the exacerbations of the chronic stage, sulphate of quinine is again useful. Apart from the attack, the more or less permanent pains that are located in the joints are sometimes successfully combated by the iodide of potassium and the ammoniated tincture of guaiacum (twenty to forty drops a day).

Externally we may employ various topical remedies during the attack. Cold water is frequently applied to the diseased joints; but, as we have already demonstrated to you, nothing is more likely to induce retrocessions.

Leeches, *loco dolenti*, used to be prescribed; but this is abandoned at the present day, since it has been observed that after their use the joints resume with difficulty their normal mobility. Narcotics, on the other hand, and atropine especially, may be applied to the diseased articulation with advantage.

Blisters are often useful, both in acute and in subacute cases. A small blister, not exceeding in size that of a *franc* (*our twenty-cent coin*), when applied to the red and swollen joint, frequently acts in the most efficient manner in the midst of a paroxysm (Todd and Cartwright). I have occasionally made use of this remedy with excellent results.

Finally, moxa is a remedy sometimes employed in this disease. We may quote in this connection the case of Chancellor William Temple, who himself applied this remedy every time he suffered an attack of the gout.

III.—*Treatment of the constitutional condition.*—The chief indication in this instance is not only to modify the blood-condition, but rather to stop the formation of uric acid in excess. This would be an ideal treatment; but how can we make it a reality? We can scarcely proceed in this manner, except in fighting a dyspepsia, where we prevent an attack by confining the patient to an appropriate diet.

Still, once uric acid has formed in the blood, we may combat the effects arising from its presence in excessive amount. The excretion of this product by way of the kidneys must be favored, and remedies possess, for this purpose, a powerful action. We should prevent deposits of urate of soda from forming in the tissues; and when these already exist, we must endeavor to dissolve them.

Empiricism has made known a group of agents that answered these indications long before the discovery of uric acid. These are the *alkalies;* and under this head are comprised:

First.—The alkali metals (sodium, potassium, lithium) and their carbonates. They have a marked effect in neutralizing the acidity of the stomach.

Second.—The organic salts (citrates, tartrates, etc.) having an alkaline base.

Third.—The phosphates of soda and ammonia, having an alkaline reaction, and a special action upon the urinary secretion.

It would be an error to suppose that all the alkalies may be indifferently substituted for one another. Let us recall, in this connection, the experiments of Claude Bernard and Grandeau, subsequently repeated by Guttman.[1] These investigators found that a gramme (15.4 *grains*) of a salt of potassium, injected into the vein of a medium-sized dog, was enough to cause its death; twenty centigrammes (*a little over three grains*) was sufficient to kill a rabbit. To obtain the same results with a sodium salt, at least three times as strong a dose was necessary.

Let us now consider the special action of each of these substances taken separately, and, beginning with the two bases which are most commonly administered, let us institute a comparison between potassium and sodium.

The salts of potassium possess a diuretic action: this fact has been thoroughly proved by Mischerlich. The salts of sodium do not possess so marked a diuretic action.

The solvent action of potassa upon uric acid is much more energetic than that of soda. It is well known that the urate of potash is much more soluble than the urate of soda. Besides, when a cartilage incrusted with urate of soda is plunged into a solution of carbonate of potassa, you observe a rapid, solvent action; if, on the other hand, it be placed in a solution of carbonate of soda, you scarcely obtain any appreciable effect in the same period of time.

Thus, *à priori*, potassa is more efficacious than soda;[2] yet the latter base is useful in those cases of gout where there is liver disease, according to Garrod.

But there is a substance little known even at the present day, namely, *lithium*, which seems in every respect to prevail over potash and soda.

This metal, discovered by Arfwedson in 1817, is present in many min-

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[1] Berliner klin. Wochen. 1865.

[2] Previous to the publication of Garrod's treatise, Dr. Galtier-Boissiére had called attention to the much greater intensity of potassa's dissolving action, compared to that of soda, in the treatment of gout. De la Goutte, p. 112. Thèse de Paris, 1859.

eral waters—in Carlsbad, Vals, Vichy, Baden-Baden, and Weilbach, in which latter town there is a newly discovered spring that is called "*Natrolithion-quelle*" (sodium-lithium spring), and contains a large proportion of this substance.

Spectrum analysis enabled Bunsen and Kirchoff to determine its presence in human milk and blood. It is not, then, a substance foreign to our organism; and if potassa exists in the blood-corpuscles, and soda in the serum, lithium is likewise found, though in very minute quantity, in the nourishing fluid of the economy.

This new agent answers all the indications we have enumerated. It has a well-marked diuretic action; it makes the urine strongly alkaline, and dissolves uric acid energetically. In this respect it is much superior to potassa, for the urate of lithia is the most soluble of urates.

Garrod performed the following experiment: into three solutions, the *first* containing five centigrammes of carbonate of lithia (*four-fifths of a grain*); the *second*, five centigrammes of carbonate of potassa; and the *third*, five centigrammes of carbonate of soda, to thirty grammes (*very nearly one ounce*) of water,—into each of these were put pieces of the same cartilage impregnated with urate of soda; at the end of forty-eight hours the lithium had completely dissolved it; the potash had only slightly acted upon it; and the soda had given an absolutely negative result.

Urate of lithia is clearly, then, the most soluble of all the urates.

What is the mode of action of the alkalies upon the blood in gout? They have no power to lessen the formation of uric acid; nor can they *dissolve* it, as various observers have supposed, for it exists in the form of urate of soda. But in rendering the tissues alkaline, they may hinder the formation of deposits; besides, the carbonates of lithia and potassa can dissolve existing deposits, a result the carbonate of soda cannot effect. Beyond this their influence would be useless, were it not for the fact that they possess at the same time a diuretic action.

This is what theory says; and now let us question therapeutical experimentation.

The alkalies, especially potash and lithia, when administered in small, in very dilute doses—for the action of water is most efficacious, and particularly when exhibited for a long period of time—possess a remarkable action in cases of gout. They postpone the paroxysms, they sometimes dissolve and diminish the depositions that have already formed, and give more mobility to the joints.

Carbonate of lithia is administered in doses varying from twenty-five to thirty centigrammes (nearly gr. iijss—gr. ivss.) for the twenty-four hours. I have prescribed it myself in forty centigrammes doses (*six-grain doses*) without producing any unpleasant effects on the stomach.

Stricker[1] has succeeded in causing tophaceous deposits in a woman to disappear, by giving her an artificial imitation of the Weilbach water, made according to the following formula:

Water charged with carbonic acid	℥ xvj.
Bicarbonate of soda	gr. iijss.
Carbonate of lithia	gr. jss.

This quantity represents the dose to be taken each day.

Schutzenberger has advised the use of water charged with protoxide of

[1] Virchow's Archiv. Bd. xxxv.

nitrogen (*nitrous oxide, monoxide of nitrogen*) containing a gramme (gr. xvss.) to the litre (2.1 pints).

Prescribing the alkalies in this way, we may be enabled to have them tolerated for several months. No serious inconvenience arises when the limits are the doses we have just mentioned.

It is also necessary to know the cases where the alkaline treatment is applicable. It is formally contraindicated:

First.—In individuals who are advanced in years.

Second.—In those whose kidneys, being more or less deranged, no longer have the power of elimination.

Third.—In those with whom alkalies disagree on account of some peculiar idiosyncrasy.

It is perhaps not ill-timed to state, in this connection, that the dangers of saturation of the blood with alkalies is much exaggerated, at least so far as bicarbonate of soda is concerned. My own personal experience is contrary to the general, accredited views in this regard. To individuals suffering with chronic rheumatism I have frequently given apparently enormous doses of bicarbonate of soda, from twenty to thirty grammes (*a little over* ʒ vijss.) in twenty-four hours, and sometimes for several months at a time; and in such cases I have never seen either profound anæmia, dissolution of the blood, or multiple hemorrhages, which might have been anticipated according to the generally received notions on this subject. But, with regard to potassa in large doses, I have not had the opportunity to directly study its effects, and I am completely ignorant of the results it may bring about.

There remain a few words to be said about mineral waters in the treatment of gout; this naturally complements the study to which we have just devoted our attention.

Generally speaking, waters loaded with saline ingredients precipitate the attacks and induce the crisis that should be averted. Certainly this is no absolute contraindication, but it is a point that the physician ought always bear in mind, so as never to be taken unawares by the effects of the treatment he has prescribed.

Mineral waters are, in general, contraindicated in patients suffering organic lesions of heart or kidneys.

Concerning alkaline springs (Vals, Vichy, Carlsbad, etc.), they seem to possess advantages at the commencement of the disease in the robust, and especially in those who have hepatic affections. But they have no power to dissolve the tophi, and they are of little benefit in chronic gout, at least when there is no dyspepsia.

Sulphur saline (Aix-la-Chapelle) or simple saline waters (Wiesbaden) are best in the torpid form in atonic cases.

There are *indifferent* waters, to make use of an expression sanctioned by German usage, whose mineral constituents are in such very small proportion (*à peines chargées*), that the real active principle is the water imbibed in large quantities. In this class we may place, from our standpoint at least, the waters of Wildbad, Töplitz, Gastein, Bath, Buxton, and Contrexeville. They are frequently very beneficial in chronic gout, In several instances we have seen Contrexeville water administered in case of a long-standing gout with tophaceous deposits, and the result seemed most favorable.

Finally, chalybeate waters (Pyrmont, Schwalbach, Spa) may also be useful in cases where iron is indicated.

We shall limit ourselves to this brief statement of the action of mineral

waters in the treatment of gout. If we were to give a critical estimate of all that has been written upon the subject both by avowed partisans of the waters, and by their adversaries, we could readily fill a volume. It is enough to say, in a general way, that on both sides there has been much exaggeration.

Let us discuss for a moment the *tonics* and *stomachics*. They possess an indirect action upon gout by modifying the condition of the stomach, by combating atony, and by reviving the strength. They are very useful in cases of asthenic gout.

The decoction of common ash (*Fraxinus excelsior*) leaves has been used with much success; it has been recommended by Pouget and Peyraud. It is prepared in the following way:

Leaves of Fraxinus excelsior (*common ash*)..... ℥ i.
Water..... Oij.
Boil for ten minutes.

Garrod has employed this infusion[1] with a certain degree of success.

Cinchona has likewise been used with advantage along with gentian, which is one of the principal ingredients of *Portland powders*.

IV.—*Treatment of the local disease, of tophi (chalk-stones), and rigidity of joints.*—We should prescribe exercise for those suffering with gout, as it tends to diminish the rigidity; this Sydenham has already demonstrated. To dissolve the tophi, it has been advised to make lotions of potash and lithia; and when the concretions are of small size and superficially seated, the skin may be punctured in order to extract them, especially when they are semifluid. But generally, when they are large, hard, and deep-seated, all operative interference is forbidden, since it frequently happens that ulcers result whose cicatrization is very difficult. Besides, it must not be forgotten that we may have a dangerous erysipelas resulting from the slightest wound in gouty patients who are attacked with kidney disease, and especially in diabetic individuals.

When ulcers form spontaneously, the rule is to avoid intermeddling.

V.—*Treatment of anomalous gout.*—It is a generally accepted and recognized fact that when gout has retroceded, especially to the stomach, we must have recourse to revulsives upon the articulations. Without questioning their utility, let me observe that there are very few authentic records sufficient to establish the efficiency of this mode of treatment in bringing back a gouty inflammation to the joints. Stimulants, cordials, and brandy are often, on the other hand, followed by readily appreciable results, and experience seems to have demonstrated their utility.

When it is a case of *misplaced* gout (megrim, ophthalmia, etc.) colchicum, administered in small doses, is indicated, according to Watson, Holland, and some other authors. But this is a question that, to us, seems far from being settled.

VI.—*Dietetic regimen.*—A gouty individual should take plenty of exercise: he should let his diet be a sober one, but in no way exaggerated, for

[1] Tanner states that the "dose is a tumblerful of a weak infusion of *F. excelsior* (one ounce of the leaves infused into a pint and a half of water), taken on an empty stomach, night and morning."—L. H. H.

otherwise the development of atonic gout would be favored. Strong beer and wines rich in alcohol must be rigorously interdicted, but he may drink light beer, moselle, and claret. He should travel; change of climate is frequently beneficial, according to the English physicians, who recommend India, Egypt, Malta, and other localities in the hot countries; but such a change does not at all dispense with the observance of a proper regimen.

Finally, mental hygiene must be regulated: the irritation so natural to this class of patients must be combated; sadness, despondency, and the preoccupation and excess of intellectual labor, must be avoided.

Treatment of Chronic Articular Rheumatism.

We have heretofore entered into so many details, that the latter part of this lecture must necessarily be abridged. It may be said that the treatment of chronic articular rheumatism is even less efficacious than that of gout; we are still less advanced in this respect, and we have not even colchicum with which to combat the most pressing symptoms of the disease.

In cases where acute phenomena supervene, the indications are almost the same as in acute articular rheumatism. Opium, sulphate of quinine, local bloodlettings, etc., are sometimes prescribed with successful results; but, in the great majority of cases, we are powerless to check the progressive course of the malady.

Large doses of alkalies, according to Garrod, are here much less powerful than in acute articular rheumatism. Still, this is the treatment in which, from my own personal experience, I have the most confidence, when in addition, quinine is employed. Besides, this is a purely empirical remedy. I have frequently prescribed from thirty to forty grammes (ʒ 7.7 to ʒ j. ʒ ij.) of carbonate of soda a day, during several weeks, with advantageous results. And, as I said when speaking of gout, I have never seen the production of any symptoms of *dissolution of the blood;* on the contrary, the patients often seem to have a certain tendency to grow stouter. By means of this treatment we are enabled to at least procure them a certain amount of relief during the febrile exacerbations of the disease.

Tincture of iodine, internally, has been highly praised by Professor Lasègue. The dose has been steadily increased from eight to ten drops a day to five or six grammes (ʒ j. gr. xviij. to ʒ jss.), given during meals, the excipient being a little sugared water, or better, a glass of Spanish wine. The drug should be continued for several weeks, and, if necessary, for several months. Its influence has never given rise to any symptoms of iodine poisoning.[1]

Arsenic has been employed by Bardsley and Jenkinson, Begbie, Fuller and Garrod of England, and by Beau and Guéneau de Mussy of France.[2] Bardsley has administered this drug especially in cases of chronic rheumatism located in the larger joints; but the other writers, whose names are given above, have exhibited it particularly in cases of nodular rheumatism. I have myself tried this remedy in the Salpêtrière, and, like Garrod, have occasionally seen arsenic produce marked amelioration, and again result in complete failure. I think, however, that I am justified in affirming that

[1] Arch. gén. de Méd. Vol. ii. 1856.

[2] Bardsley: Medical Reports. London, 1807. Kellie: Edin. Med. and Surg. Journal. Vol. iii. 1808. J. Begbie (in same journal), No. 35. May, 1858. Fuller: On Rheumatism. Second edition. London, 1860. Guéneau de Mussy: Bull. de Thérapeutique. Vol. lxvii., p. 24. 1864. Beau: Gaz. des Hôpitaux. July 19, 1864.

arsenic is without any effect—indeed, is harmful even, in the most inveterate cases of nodular rheumatism, and when the disease has set in late in life.

One of the first effects of its administration is frequently to reawaken the pains, and to make them much worse in those joints usually and most seriously implicated. Indeed, pain and swelling sometimes manifest themselves in places where they did not previously exist, even compelling us to suspend for a time our treatment. Generally, however, tolerance is established at the end of a few days, and then the dose may be steadily increased. It is advantageous, in my opinion at least, to exhibit arsenic in the form of Fowler's solution in doses of from two to six drops, and this a short time after meals, according to the method in vogue in England.

In France, while arsenic has been prescribed internally, it has likewise been employed in the form of baths by Guéneau de Mussy and Beau. In 1861, I made use of this method of treatment in the Lariboisière Hospital. Ducom, the head pharmacist of that institution, very kindly undertook the analysis of the urine of those whom I had submitted to this mode of treatment, either internally or externally. In the first case, arsenic was found in the urine after a short lapse of time; in the second the results were in all cases negative. Thus, it seems probable that these two methods do not act in the same way upon the organism, even if we admit that both are equally efficient in combating the disease—a statement that I am inclined to doubt.

There is another drug which I have employed in cases of this kind, with results analogous to those from arsenic. It is the *ammoniated tincture of guaiacum*, which first produces an exacerbation of the local symptoms, and then marked relief; the mobility of the joints reappears, and occasionally, after a certain time, the patient experiences evident amelioration.

The *iodide of potassium* has sometimes been successfully prescribed in cases of chronic rheumatism.

In chlorotic and debilitated individuals, iron and cod-liver oil may be indirectly useful by modifying the general bodily condition.

The local remedies most frequently made use of are blisters, painting with tincture of iodine, and actual cautery (red-hot points). The latter is especially useful in the partial form of chronic rheumatism.

Concerning mineral waters, Mont-Doré, Lamalou l'Ancien, Vals, Néris and Plombières have all been prescribed; most of these waters contain arsenic; can it be that the efficacy ascribed them is due to this circumstance?

We are far from having exhausted the long list of remedies that have been advocated for chronic rheumatism by various authors, or that we ourselves have tried. We have endeavored to bring prominently forward those therapeutical agents that to us seem possessed of the highest degree of efficacy; but still we must acknowledge that chronic rheumatism is a disease which, in the majority of cases, baffles all the resources of medicine.

APPENDIX.

CLINICAL IMPORTANCE OF THERMOMETRY IN OLD AGE.[1]

LECTURE XIX.

Summary.—Importance of Clinical Thermometry in General—Its Application to Senile Pathology—Central Algidity—Normal Temperature in Old Age—Axillary and Rectal Thermometry—Bodily Temperature of Old People in Pathological Conditions—Extreme Limits of the Central Temperature—Low, Medium, and High Febrile Temperatures—Danger from High Temperatures when Continued for any Length of Time—Rational Explanation of the Danger Presented by this Occurrence—Physiological Experiments—Danger from Lowering of the Temperature.[2]

GENTLEMEN:—In our previous meetings I have endeavored to make prominent the very remarkable characteristics which give a special, a unique aspect to senile pathology. I have particularly sought to set in bold relief, by means of striking examples, the assistance which medicine may derive from the methodical use of the thermometer from the triple standpoint of diagnosis, prognosis and treatment in cases where this art has to penetrate the many dangers that beset it at the clinic of the aged. To-day I purpose to continue this study, and supplement it with some new developments which, on account of the too concise form of my first lectures, I was not then enabled to present to you.

It is no longer necessary, at the present day, to expend much eloquence in defence of clinical thermometry; this method has forced its own way, and has extended almost everywhere. It was not exactly the same, however, when, in 1863, we applied clinical thermometry in the ordinary practice; still, perhaps, something remains yet to be done in order to demonstrate that this method does not belong exclusively to scientific investigation.

As you know, clinical thermometry is a physical means of exploration analogous to auscultation and percussion; but while the latter are applicable to local lesions especially, the former has to do with the fundamental phenomena of the febrile state, whose measure, so to speak, it is.[3] What then is fever? All authors to-day answer this question by Galen's definition: *calor præter naturam.* Indeed, all the other symptoms of fever may

[1] Lectures delivered by J. M. Charcot, in the Salpêtrière. 1867. (Lectures xix., xx., and xxi., are i., ii., and iii. of the Appendix, renumbered to avoid confusion.—L. H. H.)

[2] These lectures were collected together by Dr. Joffry, at that time (1867) an interne in Professor Charcot's service.

[3] Wunderlich: Verhalten der Eigenwärme in Krankheiten. Leipzig, 1868.

be wanting, but the mere elevation of animal heat remains the constant, the characteristic, the obligatory occurrence.

This is the law, the general law, and one from which old age itself is not exempt. For, gentlemen, that isolation of the organs, that want of general reaction which I have described to you in the preceding lectures, is only apparent, not real. As in children and adults, fever, or at least an elevation of the bodily temperature, occurs in old people, and frequently attains almost the same degree of intensity; but in the latter, much oftener than in the former, it may remain latent—that is to say, it may not be revealed by the external phenomena which usually accompany it. By the aid of the thermometer, however, we may seek for its manifestations in the central regions of the organism.

Gentlemen, it is especially in diseases where the temperature rises above the normal standard, that the importance of clinical thermometry can be readily made manifest; but there is a certain class of diseases, especially in old age, which give rise to reverse phenomena, by inducing actual lowering of the temperature. Now, this *central algidity* assuredly cannot be recognized except by the aid of the thermometer, which, under such circumstances, may be called upon to render the greatest service. This is a subject which is still almost uninvestigated, so to speak, but which, nevertheless, I hope will furnish us with an opportunity to learn some important facts.

I.—Before entering into the consideration of the subject, let us establish a few preliminary facts.

A.—*Normal temperature in old age.*—You know that in old age the respiratory function is decreased, as shown by a diminution in the amount of carbonic acid exhaled, by an increase in the number of inspirations, and by an appreciable reduction in the vital capacity of the lungs. It is likewise admitted that the nutritive movement of composition and decomposition is similarly lowered at this period of life, although I do not know that any decisive investigations have ever been undertaken concerning this latter point. However this may be, gentlemen, it is a remarkable fact that, in spite of these evidently unfavorable circumstances, *the temperature suffers no appreciable modification from the progress of years:* 37.2°, 37.5° (99° Fahr., 99.5° Fahr.), and rarely 38° (100.5° Fahr.) in the rectum, and now a little less or a little more than one degree (*a degree and one-half* Fahr.) above this point in the axilla. This is the normal temperature in old age, even to the extreme verge of life, according to the very numerous investigations I have made on the subject.

De Haen, in former years, and Von Bärensprung more recently state that the temperature in very old people is lower than these figures. I do not believe it to be the case except in unusual instances; three years ago, in one of these meetings, I introduced a woman who was over one hundred years old and who was in excellent health; her axillary temperature was habitually 37.4° (99.3° Fahr.), and the rectal 38° (100.5° Fahr.). Since then I have seldom found this temperature of 38° (100.5° Fahr.), representing that of the normal state, even in individuals who have attained the utmost limits of the senile period of life.

To give a *résumé*, then, the central temperature is the same in the aged as in adults; I may add that in both cases it presents the same unalterability, and that it is only raised in a slightly appreciable, but temporary manner in pathological conditions.

How may we explain the fact that the normal temperature in old age is found to be at least as high as in adult life, when in the senile state the nutritive functions are so perceptibly diminished? We must here undoubtedly adduce the condition of the skin in particular, for in old people it presents a marked impoverishment in its network of capillary blood-vessels, and at the same time its secretory activity is much below that of adult life. Probably less heat is generated in old age than in adult life, but old people do not lose so much either by the skin or by the pulmonary apparatus; and thus is established a compensation.

B.—*Axillary and rectal thermometry in old age.*—I cannot leave the subject under consideration without offering a few remarks concerning thermometry in old age, when practised in the axilla, compared with thermometry of the natural cavities, particularly the rectum. You will often hear allusions made to rectal temperature, and, indeed, the rectum is the natural cavity, which is always preferably taken as the locality for thermometrical explorations in aged individuals. I must acquaint you with the reasons which, from the beginning of my studies of this subject—that is to say, during a period of nearly seven years—have determined me in a choice that at first view might appear strange.

Gentlemen, it is easily proved and readily recognized that the temperature taken in the axilla is always lower than that exhibited after a rectal exploration; the arm-pit, from the standpoint of temperature, approximates to the surface of the body; the rectum represents the internal viscera. In truth, the difference between the temperature of these two localities in the adult, besides being usually very slight, is nevertheless almost always a (fixed) proportional difference. But this is not so in old age, where the figures representing the difference are sometimes considerable—over a degree, for example (*a degree and a half* Fahr.), and again much less, according to the most varied circumstances. Thus, in old age the central temperature alone is permanent, while the axillary temperature, on the contrary, presents extreme fluctuations, like those of the integument, although to a less extent.

But it is in the pathological, and above all in the febrile state, that we see revealed in all its completeness the relative variation between the central and the external temperature in the senile period of life. I show you a chart pertaining to a case of lobar pneumonia occurring in a woman fifty-five years of age; you see that the curve of the rectal temperature and that from the axilla, although nearly parallel in a general way, yet diverge from each other in a most irregular manner at different portions of their course. At several places you can even see these two curves presenting oscillations in opposite directions. Thus, on the fifth day of the disease, in the morning, at the very time the axillary temperature stood at only 37° (98.6° Fahr.), that of the rectum marked 40.2° (104.4° Fahr.), a difference of more than three degrees (*nearly six degrees* Fahr.). That evening the two curves approached, the difference not being over a degree (*a degree and a half* Fahr.). On the sixth day the two curves almost touched at one time, but the day after they again notably diverged. This patient had had uncontrollable diarrhœa, and at several distinct times there were symptoms of *collapse*, evidenced by well-marked coldness of the cutaneous surface. I shall, later on, recur to the signification possessed by these indications of collapse, which is a state quite often observed during the course of acute diseases in old people; let it suffice for the time being to insist upon this point, namely, that the striking disagreement we have shown to exist in the above-mentioned

case (taken as an example) between the data furnished by the axillary and rectal curves of temperature, is very frequently met with at the clinic of the aged.[1]

This is the principal reason that induces me to prefer the practice of rectal rather than of axillary thermometry; there is another—very subordinate, however—which, while it would not have been sufficient to determine us in our choice, is nevertheless not devoid of all value. At least fifteen minutes are necessary, in the aged, to obtain an exact reading from an axillary exploration. At the end of five minutes, on the contrary, the mercurial column of the instrument ceases, as a rule, to oscillate when inserted into the rectum. In view, therefore, of its facility of accomplishment, you see that rectal thermometry possesses a marked advantage over the other method, and an advantage not to be slighted in the practice of a large hospital.

I need say nothing concerning the very natural reluctance which the patients frequently manifest to the application of this form of examination, since persuasion almost always removes the difficulties arising from this objection.

C.—*Bodily temperature in the pathological state in the aged.*

First.—Extreme limits of the central temperature above and below the normal standard.

Gentlemen, there is a certain number of fundamental facts in clinical thermometry, which have been verified very many times, and which may almost be advanced as axioms. Permit me to state to you some of these all-important facts.

Whenever the central temperature, whatever the period of a disease, and whatever the character of that disease may be, rises to 41.5° (106.7° Fahr.), there is imminent danger. If it reach 41.75° or 42° (107.2° or 107.6° Fahr.), death is certain. These figures, which Wunderlich gives concerning adult pathology especially (*loc. cit.*), I affirm to hold good in all their *signification* in the aged; indeed, we may say that in old people 41° Centigrade (105.8° Fahr.), already marks a most critical situation.

If the elevation of the central temperature above a certain point indicates *per se*, and independent of the concomitant circumstances, the greatest danger, so also a fall below the normal standard when it reaches a certain point—35° for example (95° Fahr.)—reveals a most serious condition, only, however, in my judgment, so far as it affects cases of old age.

Thus, gentlemen, you notice that there are almost fixed limits beyond which, it seems, the temperature cannot pass without seriously compromising the life of the patient. Temperatures that go beyond these limits very rarely occur; they are the exception, and the forerunners, of a certain, fatal termination.

You cannot have escaped observing that clinical thermometry has already furnished us, for prognosis at least, information of the highest importance, since its *signification* is, so to speak, *absolute*. We shall frequently have an opportunity to demonstrate other equally noteworthy applications of it.

Second.—Low, medium, and high febrile temperatures in the aged.

In general terms it may be stated that a temperature rising a little over 38° (100.4° Fahr.), corresponds to a mild fever (subfebrile temperature) in

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[1] A reproduction of the chart employed in the elucidation of the preceding paragraph is not given in Professor Charcot's book.—L. H. H.

the aged as well as in adults; under 39.5° (103.1° Fahr.) it is of moderate intensity; between 39.5° and 40° (103.1° and 104° Fahr.) it is intense; and above 40° (104° Fahr.) it is exceedingly intense (*hyperpyretic* temperature).

These data, like the preceding, are as applicable in the case of the aged as in that of adults; for, with regard to the degrees of temperature that may be reached in the febrile state, the former are in no way inferior to the latter. This is a point that formerly I endeavored to establish, and which my subsequent investigations have but confirmed.

But, gentlemen—and here is an important fact for your consideration—in the comparison I have instituted between adults and the aged, from a standpoint of febrile temperature, I have spoken only of healthy individuals—that is to say, only of those free from all previous disease, from all cachexia, at the time the fever developed; for it is certain that in subjects already enfeebled, *whatever may be their age*, the temperature (even when the disease is of a nature habitually inducing intense febrile movement) cannot rise during the whole course of the malady above the medium febrile temperatures 39° to 39.5° (102.2° to 103.1° Fahr), though the case be most serious and must terminate in death. But, I repeat, and it is important to remember it—in my opinion, *this deficiency with regard to thermal reaction* is not a characteristic of the senile period of life.

In a woman seventy-five years of age, feeble and cachectic, who was attacked with lobar pneumonia, the maximum temperature at the height of the disease only once exceeded 39.5° (103.1° Fahr.). In another case of pneumonia, occurring in a woman about fifty years old—and consequently relatively young—but who had carcinoma of the uterus in an advanced stage, and a well-marked cachexia, the maximum temperature never reached 40° (104° Fahr.), but remained at 39.5° (103.1° Fahr.) or below. This patient, like the preceding, died in the *defervescence*, which is the most frequent mode of termination in cases of this kind. You recognize how analogous, in all respects, are these two observations, although taken from subjects so very different in years.

And now let us look upon the reverse of this demonstration: the three curves that I now show you[1] are tracings from three different individuals—one a child three years of age (Ziemssen); the second a man thirty-eight old (Wunderlich); and the third a woman of seventy-five, taken in this hospital. Each is a case of lobar (croupous) pneumonia. You observe that striking resemblances approximate them, and, were you not duly informed, you would with great difficulty be able to distinguish one from another. So, when Wunderlich (*loc. cit.*) affirms that, from mere inspection of a tracing of a thermometric curve, the age of the patient becomes known, since in old age the maximum is relatively at a lower point, his proposition does not seem to me to be perfectly exact. Rather, say we can recognize whether it comes from a robust individual, or, on the other hand, from one previously enfeebled.

Third.—The danger, in the aged, of high temperature continued for several days.

A third point now demands attention: a hyperpyretic temperature, 40.5° (104.9° Fahr.), may occur and yet the case not be serious, provided it remain at this point but for a very short period of time, as, for example, in a paroxysm of intermittent fever, or in abortive pneumonia. But if

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[1] A foot-note in the original says: "*We do not think it necessary to insert the three tracings in the text.*"—L. H. H.

such a temperature persist for several days almost without interruption, as occurs in diseases of a continued type—lobar pneumonia, for example—the case is very grave. What I state is based upon numerous observations that I have made concerning this subject, with reference to the pneumonia of the aged; but it seems to be established that it is nearly the same at other periods of life. However that may be, in the majority of cases of lobar pneumonia observed in the Salpêtrière, death has supervened when, during the height of the malady, the maximum temperature has several times exceeded 40.5° (104.9° Fahr.); when, on the contrary, the highest temperature has been 40° (104° Fahr.), or under, recovery was of frequent occurrence.

Let us not conclude from this, however, that all cases of pneumonia in the aged where the temperature is relatively low are mild, benignant. Far from it; you know already that many—nay, perhaps the majority—of cases of *low temperature pneumonia*, so-called, are remarkable on account of their fatality. But this is a point to which we shall recur many times in the course of the following lectures.

II.—You have seen the very practical importance of the facts that have just been adduced, but we know them only as the result of a purely empirical study, made without any theoretical prejudice whatever. The interest with which you have been inspired would certainly have been very much increased if we had attempted to penetrate into their physiological reason. Whence, indeed, these narrow limits traced upon the thermometric scale, that cannot be reached without seriously endangering life, and beyond which there is no longer any hope of saving the patient? Why does the temperature, rising to a point much short of these limits, yet persistently for several days without appreciable remission, announce a serious condition, whatever the disease it may accompany?

Such are the questions you have put yourselves. We cannot, in the present state of science, give them a definite, a rigorous solution; but we may at least hope, relying upon the data furnished by pathological physiology and experimental pathology, to discover in what direction the solution is to be found.

Let us agree, in the first place, that the derangement of the whole economy, called fever, constitutes, *per se*, a real danger when it is intense, independent of the cause that produces it. An individual is attacked with pneumonia; the respiratory functions are no more deranged than is customary in such cases; there are no complications, yet the patient dies in the midst of an intense febrile movement. The autopsy is made, and reveals lobar hepatization, which auscultation had recognized during life, but which is limited to such a small extent of a lobe of the lung that it is impossible to admit that the local lesion accounts, in this case, for the fatal issue. It is sufficient to offer this one example, for—thanks to the progress of pathological anatomy—we no longer believe, with Hoffmann, that the autopsy always discovers some great lesion in one of the organs that explains the cause of death. The constitutional condition, then, must be charged with the burden, and the febrile state in particular; but, in the midst of this almost general derangement of the organism, where is the element that is, above all others, to blame?

Here we are forced to digress.

The febrile state, with its train of symptoms—some fundamental and essential, others accessory: the rise in temperature, the acceleration of pulse, nervous derangements, and the rest—the febrile state is the result

of most complex phenomena, which may, nevertheless, be reduced to a small number of prime elements.

The starting-point seems to be a rapid metamorphosis, or, rather, an untoward combustion of the blood and tissues, which occurs almost everywhere in the organism, and is excited by the action of a morbific poison, or any other cause.

The rise in the central temperature is one of the appreciable results of the intimate chemical action constituting this exaggerated combustion.

The products of this combustion—organic waste, *urea*, and *extractives*—accumulate in the blood, and circulate along with it. Necessarily, they should be thrown out, sooner or later, by the natural emunctories, since there is no place for them in the organism.

Besides, in a case of fever with local disease, certain derangements, resulting from the abnormal functional action of the diseased organ, are superimposed upon the general disorder produced by fever. Thus, in double pneumonia and capillary bronchitis, hæmatosis is seriously impeded, and *anoxæmia*—an accumulation of carbonic acid in the blood—may occur. Or, when acute parenchymatous hepatitis (acute yellow atrophy of the liver) occurs, the materials destined to form bile are retained in the blood, on account of the rapid destruction of the secretory elements of the organ, and the result is *acholia*.

But I do not mean to enter into details, and so shall keep to the most general facts.

Whence, then, comes the danger in fever?

The rapid consumption of tissue, exhibited by more or less marked emaciation, diminution of the body-weight, and prostration of the forces, cannot be adduced, at least as the chief agent, except in cases of diseases that develop slowly. It cannot play a principal part in a fever that is rapidly evolved, such as the fever induced by lobar pneumonia, which we just now gave as an example.

The presence in the economy of organic *débris* or waste, the product of febrile metamorphosis, also undoubtedly constitutes a serious danger, in cases where excretion of these products is imperfectly performed. Indeed, we here find the conditions for an *auto-toxæmia*;[1] for these products, in certain proportions, are for the most part deleterious. This auto-toxæmia cannot occur as cholæmia and anoxæmia[2]—of which we shall soon speak—except in certain peculiar circumstances, which we shall allude to farther on in the course of these lectures. Besides, it is revealed by special symptoms which do not belong even to the most intense febrile state.

There remains, then, the rise in temperature.

Can it be that organs and tissues, when submitted for a certain period of time to the extreme temperatures we have just enumerated, undergo a sufficiently profound change in their material condition to render them incapable, at a given moment, of performing their proper functions? With this as an hypothesis, the elevation of temperature in fever is not only a result—a symptom, but is indeed the cause of derangements occasionally serious enough to induce death.

The common people, who scarcely know anything concerning fever, except the fever-heat, readily admit "that intense fever may kill the patient." This popular belief, gentlemen, should not be treated with too much disdain; for it has received recognition, to a certain extent, by such great

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[1] *Auto*, self; *toxæmia*, blood-poisoning.—L. H. H.

[2] *An*, privative; *oxus*, oxygen; *hæma*, blood.—L. H. H.

teachers in our art as Sydenham, Boerhaave, and Van Swieten. Recently it has been taken up again and, so to speak, resuscitated by many authors of good repute, among them Liebermeister[1] of Germany, and Richardson of England; and the arguments these physicians advance in its favor appear deserving of serious consideration.[2]

The best arguments have been furnished by experimentation. The central temperature can, you know, be artificially elevated in man as well as in the lower animals, and a condition closely resembling the febrile state may be thereby produced. The disorders which result when the bodily temperature is thus elevated to a certain point above the normal standard recall the symptoms of fever: thus, the pulse is accelerated and the respiratory movements are quickened; an inexpressible sense of *malaise* supervenes, and we have cephalalgia, various nervous derangements, slight disturbance at first, and extreme prostration of the forces afterward; and when, in the case of animals, the experiment is pushed farther, coma, general resolution, and finally death, supervene.

Indeed, it is known from the celebrated experiments of Blagden and Dobson, that a man may be submitted to very high temperatures, even for quite a long time, without the occurrence of any very noticeable derangement; thus, Richardson recently bore a temperature of 212° Fahr. (100° Centigrade) in a hot-air bath for nearly twenty minutes, without any discomfort.[3] But this is possible only under the express condition that, during the experiment, *the central temperature shall not rise above a certain point.* In such cases, as you know, the pulmonary and cutaneous surfaces are the seat of enormous discharges of heat, thus establishing a compensation: besides, these experiments are only possible in dry air. But, in a hot-water bath with a temperature of 40° to 44° C. (104° to 111.2° Fahr.), or a temperature no higher than 40° or 45° C. (104° to 113° Fahr.), there is great peril, inasmuch as the temperature rises—such a case is recorded by Bartels—to 39°, 40°, and even 41° C. (102.2°, 104°, and 105.8° Fahr.). In Bartels' case, and almost immediately, such grave symptoms were produced that dissolution seemed imminent.

Under conditions strikingly similar to these, that terrible accident—sunstroke—so well known to the English physicians in India, is produced; as, likewise, that analogous state popularly expressed as being *overcome by the heat.* This is fatally exemplified, in climates like ours, when armies are on the march during sultry, hot weather; the unfortunates who are thus sunstruck are at times almost literally overwhelmed—blasted, cases of this kind being recorded where the central temperature rose at the time of death to 44° (111.2° Fahr.)

But, returning to experimentation, let us consider those cases—of course we speak only of animals—where it has been carried to the utmost limit. Now, gentlemen, death always ensues, and in an almost wholly unexpected manner, when the central temperature exceeds by 4° or 5° (7.2° or 9° Fahr.) the normal standard of the animal under experiment, say 45° (113° Fahr.) for mammals. There is, as you perceive, a fatidical point for every species of animal, which cannot be attained without a fatal termination.

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[1] Ueber die Wirkungen der Febrilen Temperatursteigerung: Deutsches Archiv. Bd. i. 1866. Niemeyer Speciel Patholog. 7 Auflag. 1868.

[2] Medical Times. May, 1869.

[3] Drs. Fordyce and Blagden were able to remain with impunity in a chamber heated to 260° Fahr. (127° Cent.), and with comparative ease in one so hot that it became painful for them to touch the metal buttons of their clothing. (See Phil. Trans. 1775, pp. 111, 484.)—L. H. H.

This recalls what we have lately remarked concerning man in the pathological condition. You have not forgotten that death is certain—is a necessary sequence, indeed—when a temperature of 42° (107.6° Fahr.) is reached in the adult—that is to say, when the normal standard is exceeded by about 5° (9° Fahr.),—and a little lower than this in the senile period; and hence it is at least very likely, that in the latter instance, as in the case of animals experimented upon, the fatal issue must be largely attributed to the extreme elevation of the central temperature.

But what is the mechanism of death? The experiments of Claude Bernard,[1] and Calliburcés, repeated by Panum, establish the fact that it is the heart which especially suffers in these cases; at first excited in its functional activity, it finally ceases to beat when the temperature reaches 45° (113° Fahr.). The organ presents no gross lesions, but its tissue has undergone profound changes, muscular rigidity being produced comparable to cadaveric rigidity, and movements cannot possibly be induced, even for a time, by subjecting it to the influence of excitants.

A marked alteration in the constitution of the blood likewise occurs: sometimes it is very fluid; sometimes, on the other hand, coagulated. According to Richardson, the first occurs in cases of very sudden death; the second when death has been postponed for some time after the commencement of the experiment.

In this connection, let us not forget that, in pathological conditions occurring in man, where death has rapidly ensued, and has been preceded by a considerable rise in temperature, the blood has sometimes been found in a state of extreme fluidity, and sometimes, on the contrary, coagulated within the cavities of the heart. Boerhaave thinks that coagulation of the blood in the vessels is one of the causes of death in fever, and quite recently Weikart has endeavored to prove that this, indeed, was the attributable cause in cases where the temperature rose to about 42° (107.6° Fahr.).

All the preceding facts have reference to cases wherein death followed extreme elevation of the central temperature. In those in which the febrile heat was maintained for a longer or shorter period at a lower, although still relatively high point, we can no longer appeal to the data furnished by experimental physiology, for experiments have never been made in this particular direction. But we may, nevertheless, adduce certain facts that tend toward making us admit that even here elevation of temperature may *per se* induce serious symptoms, and itself be the actual danger.

Let me first observe, with Liebermeister, that most febrile affections, whatever may be their nature, in which the temperature is maintained at a high point during a certain time, and in a more or less permanent manner, possess an almost constant and common character. In such cases as these, certain organs present, at the autopsy, parenchymatous changes that have sometimes been designated by the name of *steatosis*. The liver, the muscles of organic life, the kidneys, and the heart especially, undergo this degeneration. I shall only cite, in this connection, the more or less marked softening which the last-named organ may undergo in typhoid fever—according to Louis, and in typhus—according to Stokes. These lesions are always accompanied, during life, by symptoms of cardiac weakness—asystolism. Can we ascribe these changes, especially those of the heart and the consequent derangements, to the permanence of the high temperature? We might be led to admit it from the action that extreme temperatures exert on the cardiac tissue.

[1] See the recent investigations of Claude Bernard on the influence of heat upon animals: Revue Scientifique. Nineteenth year. No. 8. 1871.

It is well known, too, that great acceleration of the pulse, which is one of the most dangerous symptoms in cases of this kind, is, to a certain extent proportional to the rise in temperature.

But here is an argument that bears rather more directly upon the question; it is derived from the undoubted advantages of antipyretic treatment adopted in the case of acute diseases accompanied with high temperature. Now, what is at the same time the most prominent, the most constant, and the best-established result of the various remedies employed in this method? It is a more or less well-marked lowering of the central temperature. for a longer or shorter period of time, even at the acme of an intense febrile movement. Thus do digitalis and veratrum act in pneumonia, and sulphate of quinine in acute articular rheumatism; in this way, too, act the prolonged and frequently repeated cold baths, recently employed with so much enthusiasm by the Germans in their treatment of typhoid fever, and which seem to have produced results that are certainly worthy of attention (Brand, Jurgensen, Liebermeister, and Gerhardt).

From the preceding, we think we can offer this conclusion—not, indeed, as the result of a rigorous demonstration of its truth, but at least as a very probable hypothesis, namely: *a rise in the central temperature, in fever, itself constitutes a danger.*

Let us leave for a moment the discussion of fever and rise of temperature, to consider a thermometric change in the other direction; let us see, in other words, why a fall of temperature to a certain point below the normal standard is almost necessarily followed by death. Has the cold developed in such cases within the organism a positive action upon organs and tissues capable of diminishing the degree of energy of organic activity necessary to life? Gentlemen, there can be no doubt but that such is actually the case.

When an animal is, under certain circumstances, submitted to the action of a low temperature, in a state of complete inanition, poisoned by certain substances—opium, ammonia, hydrocyanic acid—his body covered with an impermeable jacket, as in the experiments of Fourcault and Edenhuisen, the central temperature always falls, and at the same time the respiratory movements become feebler and feebler; the absorption of oxygen and the exhalation of carbonic acid are diminished.[1] If the experiment is carried farther, the temperature falls to a point below which death will ensue. Now, what occurs in these various circumstances? It may be said the animal *dies of cold.* He dies of cold, since artificial heat always retards the fatal termination—indeed, even permitting, in the most favorable conditions, a complete restoration to life.[2]

Thus, gentlemen, theoretically as well as experimentally, heat and cold, when carried to a certain extreme in the organism, are elements of prime importance, and elements that must be taken into account, not only to establish a prognosis in a given pathological condition, but also to regulate the administration of therapeutic agents.

But I perceive—a little late, perhaps—that the digression I entered into has led me far astray, and that it is time to return to senile pathology.

It is not enough to have recognized that, in old age, the elevation or fall of the central temperature to a certain point has the same significance as at the other periods of life; we must actually establish the fact that oscillations of temperature carefully recorded day after day, hour after hour,

[1] Valentin: Arch. für physiol. Heilk., p. 433. 1858.
[2] Brown-Séquard: Soc. de Biologie. Vol. i., p. 102. 1849.

in the form of graphic tracings, furnish constant and characteristic types for every disease in the aged as well as in adults, as I have already demonstrated in case of the latter. For it is upon this very circumstance that a diagnosis of febrile diseases by means of temperature is founded, a method that has received much attention at the present day. You will see, as I have already indicated, that these specific types do not undergo noteworthy change, notwithstanding the age of the patient; in their essential features they are in the aged what they were in the adult. In our next lecture we shall endeavor to bring additional proof in support of this assertion; we shall then have an opportunity to review, from this particular point of view, the diseases which produce fever in the aged.

LECTURE XX.

Summary.—Thermal Characteristics of Febrile Diseases in Old Age—Febrile Diseases of the Continued Type—Febrile Diseases of the Remittent Type—Febrile Diseases of the Intermittent Type.

Rapid Rise in Central Temperature, at the Time of Death, in Certain Diseases of the Nervous Centres—Tetanus—Epilepsy—Hysteria—Cerebral Hemorrhage and Softening—Epileptiform and Apoplectiform Attacks.

GENTLEMEN:—At the close of my last lecture I told you that the fundamental characteristics of thermometric curves are not strikingly altered in old age. Let me justify this assertion by taking for examples some of those febrile diseases, called *typical*, that occur in old people. These diseases may be arranged in three groups, according as the febrile type is continued, remittent, or intermittent.

A.—THERMIC CHARACTERS OF THOSE FEBRILE DISEASES OF OLD AGE DENOMINATED TYPICAL.

First.—Febrile diseases of the continued type.—These are much less numerous than in the adult; the eruptive fevers do not occur. Nevertheless, I have occasionally seen small-pox in the Salpêtrière. In the majority of these cases it assumed the hemorrhagic form attended with collapse. The patients usually suffered a remarkable lowering of the central temperature, a true *algidity*, a condition quite worthy of your attention, and of which I purpose to speak in the next lecture.

The principal disease of this group is *lobar* (*croupous*) pneumonia, which, notwithstanding the contrary statement of many authors, is more frequently observed in old age than broncho- (*catarrhal*) pneumonia.

a.—The disease is usually ushered in by a chill—the statement that the aged seldom have a chill is wrong. At the same time the extremities become cold. This period, the *pyrogenetic*, is marked by an *abrupt rise* in the thermic line. The next day the temperature rises to 40° (104° Fahr.), for example. This characteristic is of itself amply sufficient to distinguish this disease from those of the following (second) group, where the rise is slow and progressive.

b.—At the time when the disease is at its height, the temperature, having attained a certain point, remains nearly stationary for a number of days. We must, nevertheless, note the occurrence of diurnal variations, quite narrowly limited and not exceeding a degree (*a degree and a half* Fahr.). In its development this part of the curve exhibits, at times, a progressive tendency to rise, and then we have to deal with a serious case; again, there is a tendency toward a fall in temperature, occasionally predicting a favorable termination, but only when the other symptoms, taken collectively, are not aggravated.

The regularity of the curve may be altered through various circumstances; it is enough, for the present, to mention the modifications resulting from the administration of drugs.

c.—The third period is marked by a different curve, according as the termination is to be fortunate or fatal.

In the former case you observe a period of healthy defervescence, occasionally preceded by a temporary exacerbation of all the symptoms attended by an abrupt ascent of the curve. This corresponds to what the ancients called the *perturbatio critica*. This aggravation of symptoms is of short duration in cases that are to recover, lasting only a few hours. Defervescence then occurs, and usually runs a very rapid course; the curve descends with a single stroke, thus recalling inversely the rapid variations in temperature that marked the commencement of the disease. In this sudden fall we often observe the occurrence of *subnormal* temperatures, attended by symptoms of *collapse*, which we shall consider later on. Soon, however, the curve regains the normal level, and stays so permanently. Convalescence has begun, in spite of the persistence of local symptoms.

It is generally after the beginning of defervescence that we notice those critical phenomena which so attracted the attention of the ancients. Here thermometry shows itself superior to the older method of observation, since the final lowering of the temperature precedes the appearance of the critical phenomena. In passing, let me remark that, in old age, these occurrences are less frequent than in adult life. In very rare instances they consist of epistaxis or sweats, but generally they are exhibited either by diarrhœa, or again, by copious passages of turbid urine.

When there is to be a fatal termination we sometimes notice a rapid rise in the temperature, which we have occasionally seen to continue increasing even after death. This is in the vast majority of cases what generally occurs in the adult. And it is also what we observe in healthy old people; whereas, in enfeebled aged individuals, death commonly occurs in the defervescence. In this ominous defervescence the temperature does not generally fall as low as in favorable defervescence. In our next lecture we shall see by what characteristics we can affirm whether the termination is to be in death or recovery, in cases where defervescence sets in at the decline of lobar pneumonia.

Second.—Febrile diseases of the remittent type.—Here lobular or catarrhal pneumonia assumes the first rank; and indeed, as we have already said, it is much rarer in old age than has been supposed.

In the pyrogenetic period the ascent is slow and jerking. In the acme the daily oscillations are quite extensive, usually greater than a degree (*a degree and a half* Fahr.); lastly, there are no critical phenomena at the moment of defervescence.

Concerning typhoid fever and acute catarrhal phthisis, the most prominent and important diseases of this group in adult life, we may say that they are almost wholly wanting in old age.

Third.—Febrile diseases of the intermittent type.—In the Salpêtrière, paludal fever is very rare; indeed, I am not absolutely positive of ever having seen it in this hospital. Symptomatic intermittent fevers, from diseases of the urinary or biliary passages, are, on the contrary, very frequently met with in this institution. These symptomatic fevers can be distinguished from paludal (*malarial*) intermittent fevers (*ague*) by means of certain characteristics, and from their frequence they seem to deserve so much attention that I shall devote some of our future meetings to their consideration.[1]

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[1] The recent thesis of Dr. Magnin contains the substance of these lectures. De quelques Accidents de la Lithiase biliaire. Paris, 1869. See the Second Appendix. (*There is no "Second Appendix" to the book.*—L. H. H.).

B.—The Rapid and Considerable Rise of Central Temperature Occurring at the Time of Fatal Termination in Some Diseases of the Nerve-Centres.

Until this moment we have only been concerned with febrile diseases; but in the course of other diseases we may, at a given moment, witness an abrupt and very great rise in the central temperature. Let me dwell a few moments upon the study of this phenomenon, that is yet hardly known, although meriting attention, were it only on account of its prognostic importance.

Deep coma, sometimes, though rarely, preceded by delirium, a very great increase in the pulse-rate, contracted pupils, occasionally tonic or clonic convulsions, the rapid development of sloughing sores (*eschars*) about the anus—such are the phenomena that usually accompany the elevation in the central temperature under discussion. It occasionally reaches 41° or 42° (105.8° or 107.6° Fahr.), or even higher; and it may even go on increasing for a few moments after death.

It may be asked, whether in cases of this kind we must rely on the ordinary mechanism of fever to account for the production of these high temperatures. Wunderlich says, though rather vaguely, that "then the products of tissue-metamorphosis do not appear in excess in the urine." In two cases of tetanus observed in horses, Senator says that only a faint trace of urea was found in the urine.[1]

What may be said is that, in all these cases, the nervous system is profoundly involved. Diseases such as *tetanus*—traumatic or non-traumatic—and *epilepsy* of the variety called *essential*, are examples that furnish types of this *terminal* elevation of temperature.

These two diseases are accompanied by tonic spasms, and it is generally after repeated attacks that the fatal rise in temperature occurs; but still we cannot adduce muscular contraction as the cause of this considerable increase in heat, for in ordinary cases of tetanus or epilepsy the most intense convulsions produce but a comparatively slightly marked elevation in temperature (A. Monti: "Beiträge zur Thermometrie des Tetanus." Centralblatt, No. 44, 1869). The thermometric scale rarely marks 39° in these cases (102.2° Fahr.). On the other hand, in cases where this great terminal elevation has been observed, the convulsions have often long since ceased, and given way to a more or less profound coma. Some cases of *hysteria*, or at least of *hysteriform affections*, have been recorded, accompanied or not by convulsions, which terminated fatally, and presented this final elevation of temperature.

One general fact is, that in no one of these cases, either in nerve-centres or in viscera, was there any recent material change capable of accounting for the terminal symptoms and rise in temperature.

You know that in the *general paralysis* of the insane we quite frequently observe attacks that are at times apoplectiform and attended by more or less lasting paralysis, and again are of an epileptiform nature with subsequent coma.

The investigations of Westphal show that the temperature rises to about 39° (102.2° Fahr.) fifteen minutes or an hour after these attacks,

[1] Quite recently in a case of tetanus in a man, where the central temperature was elevated and death followed, Senator found the excretion of urea below the normal standard.—Virchow's Archiv. October, 1869.

whether convulsions have occurred or not. It falls rapidly if the case is a favorable one; but when death is to ensue, it continues high and even goes on rising. At the autopsy no other lesions are found save those of diffuse peri-encephalitis (*meningitis*). In some cases, however, the existence of spots of pulmonary induration has been determined, but by far the greater number of cases have no cause assignable for the terminal symptoms.

I should not, gentlemen, have recalled these facts, which belong to the domain of pathology of mind diseases, were it not that they have their analogues in diseases more particularly belonging to senile pathology. We have, in fact, in these wards a great number of patients who, for a longer or shorter period of time, have been attacked with hemiplegia following upon cerebral hemorrhage or softening. Now, among these it is not uncommon to see the development of a disease marked by *apoplectiform* or *epileptiform* attacks, recurring with more or less frequency. The greatest analogy, both as to form and consequence, exists between these attacks and the corresponding symptoms in the paralysis of the insane. Death may occur in, or subsequent to these attacks, as in diffuse peri-en-

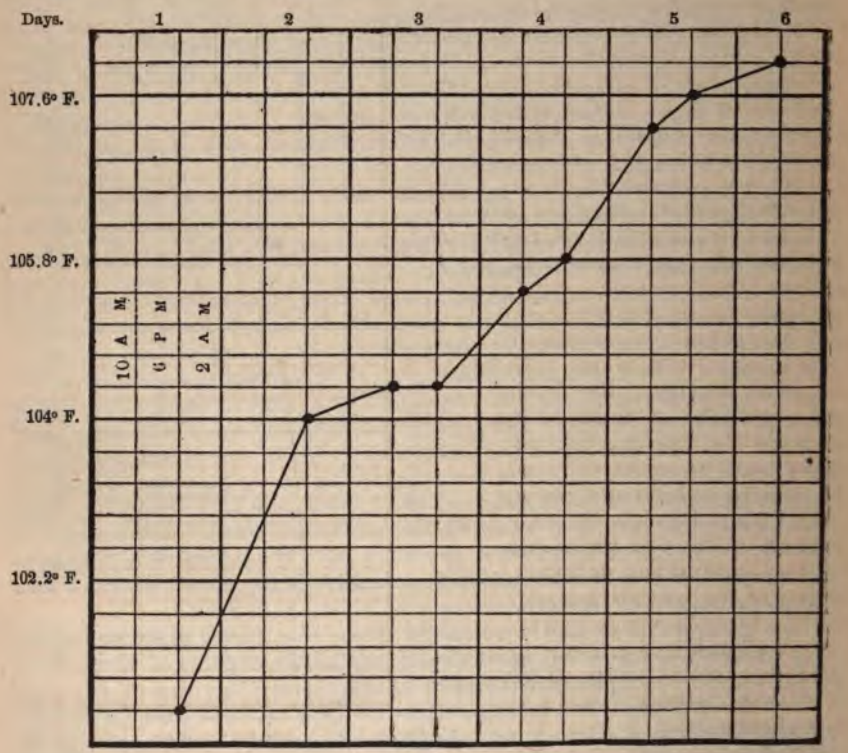

FIG. 26.

cephalitis, and in such cases we always observe a rapid and well-marked elevation of the central temperature. Now, however carefully the autopsy may be made, it is impossible to discover, either in the nerve-centres or in the viscera, any *recent* lesion that is capable of explaining the grave symptoms that resulted in dissolution.

We only find old lesions (foci of hemorrhages or softenings) that account for the hemiplegia, and secondary degenerations of the mesocephalon and brain-substance, the result of changes in the hemispheres. I think, by way of exemplification, that it may not be useless to exhibit two plates relative to cases of this kind.

The first is that of a woman, thirty-two years old, suffering incomplete right hemiplegia, dating from infancy, with the atrophy and contraction of the paralyzed limbs generally observed in such cases.

After an attack much severer than usual, she was brought to the infirmary. The rectal temperature was 38° (100.4° Fahr.) on the day she entered. The attacks became subintrant, occurring about one hundred times a day; they were separated by a coma which grew deeper and deeper; sloughing sores rapidly formed on the buttocks and the patient died on the sixth day. The central temperature increased every day, reaching 42.4° (108.32° Fahr.) the day she died (Fig. 26). At the *post-mortem* examination there were found, on the left side of the brain, quite a large depression, an extensive yellow patch, and atrophy of all the hemisphere on that side. There were no recent lesions of the nerve-centres or viscera.

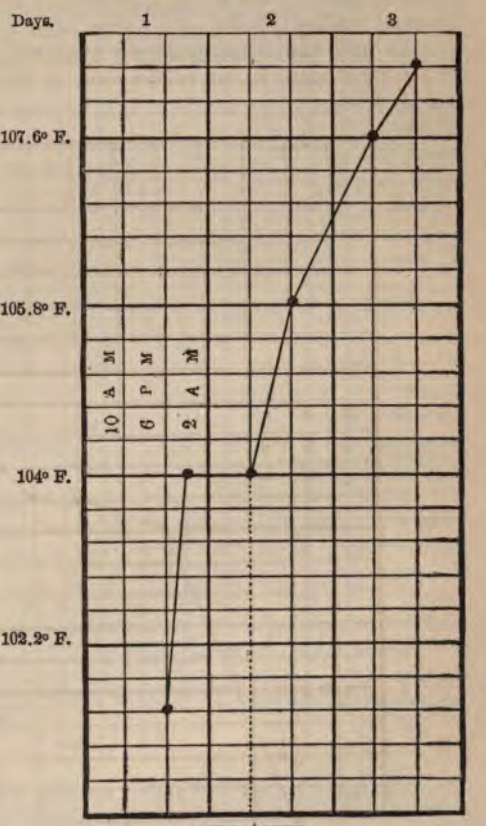

FIG. 27.

The second case is that of a woman, aged sixty-one, with right hemiplegia, the result of a cerebral hemorrhage of two years' standing. This patient had already experienced several, though generally quite mild epileptiform attacks. One day an intense and prolonged epileptiform attack occurred, followed by a condition similar to that of apoplexy. Two hours after the beginning of this, the rectal temperature was 38.6° (101.48° Fahr.); five hours later it was 40° (104° Fahr.). Next day, *notwithstanding the convulsions had ceased*, the temperature was 41° (105.8° Fahr.); and the day after that—the day she died—it reached 42.4° (108.32° Fahr.). (See Fig. 27.)

At the autopsy were found two ochreous foci—one in the corpus striatum, the other deep down in one of the convolutions. But there was no recent lesion that could be held accountable for the symptoms.

A few moments ago, I demonstrated the clinical utility of thermometry in cases of this kind. I shall show you that the results it gives furnish

most important indications not only for prognosis, but also for diagnosis. But I have not finished the enumeration of the diseases of the nerve-centres, in which ultimate high temperatures are met with. I shall now consider the diseases where there are recent lesions, and begin with those which are traumatic.

Since the celebrated case of Brodie's, where the cervical marrow was crushed on account of luxation of the fifth and sixth vertebræ, and where the temperature was 43.7° (110.66° Fahr.), quite a large number of similar observations have been published (recently by Fischer, Naunyn, and Quincke). In a lecture delivered in England several years since, Brown-

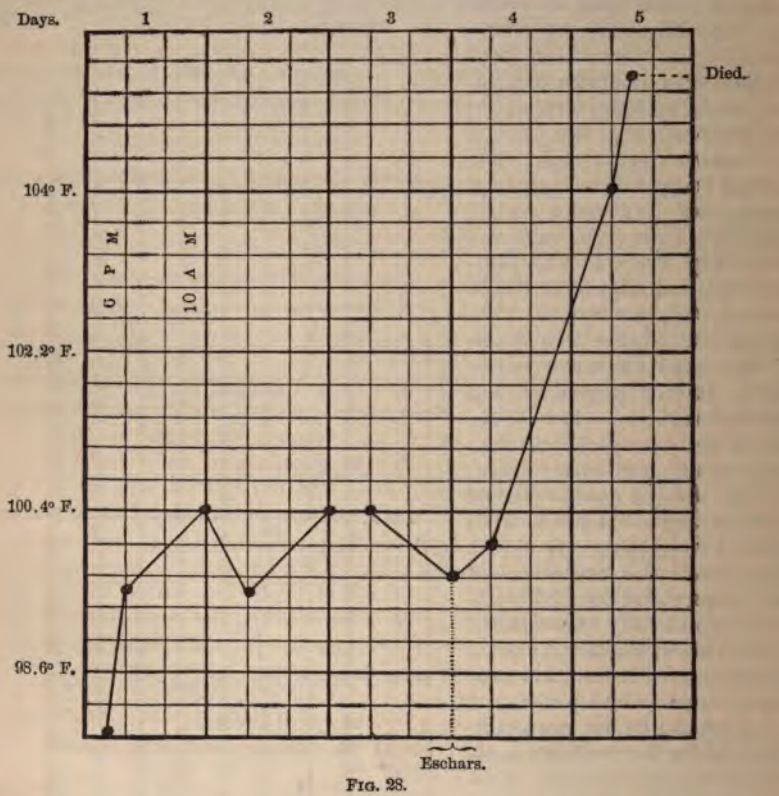

FIG. 28.

Séquard had already collected records of some of these cases, and their analysis led him to the important conclusion that elevation of temperature presupposes a grave medullary lesion, whereas when the cord is merely irritated we observe coldness. Dr. Fischer has quite lately drawn the same distinction.

Moreover, this is not the case with lesions of the cord alone. Billroth has seen death rapidly ensue from a fissure of the cranium without any external wound: the central temperature rose to 40.9° (105.6° Fahr.).

We find these same characteristics in serious lesions of the brain, non-traumatic in origin, such as hemorrhage or softening. I have proved by repeated observation, that, in general, death in these cases, as in the former,

is preceded by an abrupt rise in temperature, sometimes reaching 40° and 41° (104° and 105.8° Fahr.). Thermometrical examinations here aid in establishing our prognosis, for, as a general thing, the temperature does not exceed, or at least only by a very little, the normal standard in case the apoplexy arises from cerebral hemorrhage or softening of recent date, when no phlegmasia complicates ; so that an abrupt rise in temperature, in such cases as these, is one of the most ominous signs.[1] I have, till the present time, met with only a very few exceptions to this rule.

As I have said, there is a thermic characteristic by means of which apoplexy (with or without convulsions) arising from a recent lesion—hemorrhage or softening—may be distinguished from those apoplectiform or epileptiform attacks occasionally supervening upon old cases of hemiplegia. Indeed, in the latter case, the temperature is always more or less elevated during the first hours of the attack,[2] whereas in true apoplexy, caused by cerebral hemorrhage or softening of a recent date, there is at the beginning an almost constant lowering of the temperature below the normal standard (see Fig. 28).

What is the physiological reason for the phenomena we have just described? Wunderlich and Erb, the first who called attention to this subject, assume for the requirements of the case that certain parts of the nervous system possess, normally, a restraining influence (*inhibitory action*) upon the centres for calorification. Now, if these supposed moderating centres are profoundly injured, the result will be that the chemical acts productive of heat will be accomplished unrestrainedly—inordinately—and hence an abrupt and often enormous rise of temperature will be the consequence.

But where is the seat of these restraining, inhibitory, regulating centres? Recent experiments of Tscheschichin appear to give an answer to this question.[3] If the cord be cut transversely at different localities, the members become hotter as a result of the vaso-motor paralysis that follows such section ; but there is a simultaneous cooling of the central portions, arising partly from the discharge of heat, and partly from the cardiac weakness, the result of the peripheral accumulation of blood. But, if a section be very carefully made at the level of the junction of the protuberance (occipital) and the medulla oblongata, the central temperature almost immediately thereafter rises, to attain, at the end of two or three hours, a very high point. The pulse-rate and respirations are at the same time increased. From these results the experimenter has concluded that there are in the encephalon, above the point mentioned, moderating (inhibitory) centres whose paralysis induces an exaggerated and ungovernable production of heat.

It would be desirous to have a repetition of these experiments. Should the results be confirmed, they could be usefully combined with clinical facts.

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[1] Mémoires de la Société de Biologie. Vol. iv., Fourth series, p. 92. 1867.

[2] The comatose condition that terminates the majority of cases of cerebral tumor is likewise accompanied by a sudden elevation of the central temperature. I have seen this occur very frequently, and a case reported by Ladame also corroborates it.—Symptomatologie und Diagnostik der Hirngeschwülste, p. 164. Wurzburg, 1865.

[3] Deutsch. Archiv, p. 398. 1866.

LECTURE XXI.

Summary.—Central Algidity—The Disagreement that may Exist Between the External and the Deep Parts—Lowering of the Central Temperature in Chronic Diseases—Cancer, Anæmia, Diabetes, Phthisis—Lowering of the Temperature in Acute Diseases—Effects of Drugs and Poisons—Physiological Experiments—Septicæmia, Cholæmia, Uræmia—Cardiac Diseases—Pleurisy, Pneumothorax, Peritonitis—Diseases of the Spinal Cord—Clinical Significance of Collapse—Algid Pneumonia—Pestilential Diseases.

GENTLEMEN:—Until the present moment we have simply discussed some of the pathological states wherein the temperature rises above the normal standard; but it is not rare, in old age especially, to observe the reverse phenomenon during the course of certain diseases—that is to say, an actual diminution of the central temperature. To this subject I wish to-day to call your attention.

While there are diseases wherein the febrile state constitutes an essential, obligatory characteristic, there are none which *necessarily* induce, in all their course, a lowering of the temperature below the normal standard; so that *central algidity*—for thus shall I designate the present subject—usually appears in the history of disease only as an episodic occurrence—a symptom that in the vast majority of cases is transitory. Let me add that frequently the gravest significance is betokened by this sign.

Perhaps many of you, thinking of one of the most prominent symptoms of Asiatic cholera—the cadaveric coldness of the extremities—find that the proposition we have just enunciated is over-absolute. But thermometry does not stop with external phenomena. Now, what does it teach us in Indian cholera? In the algid stage it is true that the temperature falls below the normal standard in a very remarkable manner—less, however, than the sensation experienced by the hand of the observer would lead him to suppose. The hands and feet, in the severest cases, give a thermometric marking of 35°, 31°, 29° (95°, 87.8°, 84.2° Fahr.); but during this period, contrary to expectation—contrary to all the information furnished by axillary thermometry—the central temperature does not usually vary. This is a fact whose demonstration I commenced at the time of the epidemic of 1866,[1] and which, later, was confirmed, in a most striking manner, by investigations, undertaken on a grand scale by Guterböck of Germany, and my colleague Lorain, of France. In seventy-four cases collected by the latter author, the rectal temperature fell to 35° (95° Fahr.) four times, and only once to 34° (93.2° Fahr.); in five cases it reached 40° (104° Fahr.); while in all the remaining cases it oscillated between 37° and 38° (98.6° and 100.4° Fahr.). Thus, in cholera itself, until now considered as the type

[1] Note sur la Température du Rectum dans le Choléra Asiatique: Comptes Rendus des Séances et Mémoires de la Société de Biologie. Vol. xvii., p. 197. Paris, 1865, 1866. Lowering of the Temperature in Disease, by Cherbach: Thèse de Strasbourg. 1869.

of algid diseases, the coldness is all external, and does not involve the central portions of the organism.

There is also quite a number of pathological conditions besides cholera, where we notice a disagreement between the external temperature and that of the deep parts. In certain modes of death, for example, it is not unusual to find the thermometric record from the rectum attaining hyperpyretic limits, while the extremities remain cold. And, let me remark, the reverse never occurs. The increased heat of the external parts does not exceed that of the central temperature, except, perhaps, in the one case of local inflammations. John Simon concluded from his experiments that the inflamed region is a focus wherein the temperature may rise some tenths of a degree above that of the arterial blood which flows to the part. Recent experiments of Weber have in fact confirmed this.[1] When the case is no longer one of inflammation, but rather of simple neuro-paralytic hyperæmia, as occurs in certain forms of paresis, or in the course of certain febrile diseases—pneumonia,[2] for instance—the temperature of the hyperæmic parts is always lower than that of the deeper parts of the economy.

I.—But, to return to central algidity. I have already said that it very rarely manifests itself in a persistent manner in the course of a disease. The few examples of this sort that we may cite concern chronic diseases. Cancerous affections should here be placed in the foremost rank, but only under certain conditions—when, for instance, we have emaciation, atrophy, and inanition going on to actual marasmus. Hence, it is in gastric and hepatic cancers that we especially notice this central algidity. With these exceptions, the temperature of the most varied forms remains about normal, although there may be a slight increase in the central heat. This is a fact of which we became assured this year, from the observation of quite a large number of women afflicted with cancer either of the breast, uterus, or face.

The same conditions of inanition and marasmus may be met with in other diseases than cancer; in this connection we may mention profound anæmia, diabetes, and phthisis in certain cases. The temperature may remain lowered, often for quite a long period of time—at 36° (96.8° Fahr.) or under—which, however, does not prevent the temperature from undergoing, at moments, and especially toward evening, a relative rise of one degree or more (*one and eight-tenths or over*, Fahr.), which is now and then evinced by a chill. Under such circumstances the body-weight progressively and rapidly diminishes, while the temperature continues to fall until dissolution occurs (*Zehrfieber:* O. Weber).

It is undoubtedly on account of inanition that a more or less enduring fall in temperature has been quite frequently (Wolff) observed in subacute and chronic mania, with symptoms of depression, chiefly melancholia, attended with stupor. But the interpretation we offer cannot be applied to all cases of this kind. Quite recently, indeed, Dr. Löwenhardt, of Sachsenberg, has reported two cases of insanity where the rectal temperature reached the almost incredible points of 31°, 32°, and 32.5° (87.8°, 89.6°, and 90.5° Fahr.), persisting during several weeks, while nutrition did not

[1] Recent observations of Jacobson and Bernhardt (Berlin Centralblatt, No. 19, 1869), of Landieu (*id.*), and Schneider (*id.*, No. 34, 1870), seem to confirm the opinion of Hunter, and consequently invalidate the results obtained by Simon, Billroth, and O. Weber.

[2] R. Lépine: De l'Hémiplégie Pneumonique. Thèse de Paris. 1870.

appear to be affected in any noteworthy degree. One of these patients was excitable, the other erotic, and both took sufficient nourishment.[1]

II.—Central algidity is especially interesting, as an incident occurring in the course of acute diseases.

First, however, let us seek for the principal conditions inducing, in such a case, this lowering of the temperature.

A great number of substances employed as *drugs* have the effect of causing a more or less profound depression of the central temperature. And it is especially when they are taken in large quantities, approximating to toxic doses, and principally, too, when exhibited in the course of the febrile state, that the action of these drugs is most energetically manifested. In this way act digitalis, sulphate of quinine, calomel, and even alcohol.

But when they are given under ordinary physiological conditions, they must be carried to relatively enormous doses before they produce a lowering of a few tenths of a degree. This, for example, is the case with alcohol.[2]

When these self-same substances are taken in toxic doses, they induce, in the majority of cases, a considerable lowering of the temperature, which perhaps contributes in great measure, as we have already remarked, to bring about a fatal result. Among other substances that produce this result we may mention chloroform, ether, alcohol, opium, belladonna, nicotine, phosphorus, and the majority of acids—sulphuric, oxalic, hydrocyanic, etc.[3]

It is interesting to note that, while a variety of drugs and poisons have the power of lowering the central temperature, a very limited number of agents, on the other hand, possess the opposite potency. We can scarcely mention more than three or four that have the property of increasing animal heat.

These are strong black coffee, tea (Lichtenfels and Frölich), but to a less extent musk (Wunderlich), and finally, curare, which, according to Voisin and Liouville, produces an actual febrile state in which the central temperature may rise to 40° (104° Fahr.).

This is the appropriate occasion for a study of the variations in temperature produced by the action on the organism of morbid poisons, and animal or vegetable substances in process of putrefaction.

The majority of putrid matters introduced into the blood in physiological experimentation have the effect of elevating the central temperature and causing an attack of fever, accompanied by chills, acceleration of pulse-rate, loss in body-weight, etc. The experiments that recently have been repeated so many times by Billroth, Weber, Fischer, Bergmann and a host of other investigators, give almost the same results; and fever is induced, not only by the injection of putrid materials, but also by the products of tissue-metamorphosis congregated, for example, in an inflamed wound, even though there be no trace of putrefaction.[4]

According to modern research, we know that traumatic fever is produced

[1] Allgemeine Zeitschrift für Psychiatrie. Bd. xxv., p. 685. Berlin, 1868.

[2] Sidney Ringer: The Influence of Alcohol, etc. Lancet, October 25th, August, 1866, p. 208. H. C. Gell and Sidney Ringer: The Influence of Quinine on the Temperature of the Human Body in Health. Lancet, October 31, 1868.

[3] Brown-Séquard: Société de Biologie. Vol. i., p. 102. 1849. H. Wyss: A Case of Poisoning by Sulphuric Acid. Archiv der Heilkunde. Heft 2. 1869. Magnan: Cas d'Empoisonnement par l'Alcoöl. Gazette des Hôpitaux. No. 82. 1869.

[4] Venom seems to act in a similar manner.—Case of snake- (cobra) bite, successfully treated by sucking, liquor potassæ, and brandy. John Shortt: Lancet, April, 16, 1870.

in an analogous manner. The fluids thrown out from the surface of wounds, and loaded with products formed by destruction of tissue, penetrate, by diffusion, through the walls of the lymphatics and veins, thus commingling with the blood. Thus it is they develop the febrile state by reason of this *pyrogenetic* power, the existence of which has lately been proved by experimentation.

Experiments have likewise proved that the blood of a feverish animal, when injected into the veins of a healthy animal, induces fever. And even a rather profuse bleeding, which in a healthy individual has had the effect of producing a fall of temperature for a short period of time, may subsequently produce a true febrile state. On account of the diminution of tension occurring in the vascular system as a result of the loss, the products of the normal metamorphosis of tissue suddenly enter the circulation in great abundance, and there comport themselves after the manner of pyrogenetic substances. Such, at least, is the interpretation given by Bergmann and Frese, the investigators who conducted the experiments.[1]

It seems then to be established, that the majority of septic substances contained in pathological fluids have the effect of causing fever. But it is equally true that a certain number of this kind of substances have a diametrically opposite action upon the organism. Thus, for example, in the experiments of Weber and Billroth, the injection of putrid animal matter or putrid pus has very often determined a marked diminution in the temperature ; commonly this is followed in a short time by a more or less intense febrile condition, now persistent in character, and again, on the contrary, progressively aggravated until death occurs, which, in the latter instance, generally ensues very quickly.

It is difficult to absolutely predetermine what putrid matter will, when injected into the blood, produce fever, and what, on the other hand, will induce central algidity ; for, under the name of putrid or septic matter, are comprehended substances of the most varied chemical composition. It is, at least, very probable that the same substance which, taken at a given period of putrid fermentation, would cause fever, might, if used at a more advanced stage of the work of decomposition, induce the opposite result—algidity. The principles whose presence in the putrefying matter may be determined by chemistry vary, of course, according to the nature of the substances whence they originated, and according to the different stages of putrid fermentation. Now, among these principles are some which, when separately injected into the blood, have the effect of lowering the bodily temperature. Such, according to the concordant experiments of Billroth, Weber, and Bergmann, are ammonia carbonate, butyric acid, ammonia hydrosulphate, and hydrosulphuric acid (*sulphuretted hydrogen*). If, then, there is a predominance of these agents over the pyrogenetic substances in a fluid, we can readily understand what effect will be produced upon the organism by the injection or absorption of such a fluid.

These data, taken from experimental pathology, are, we think, the key to a certain number of apparently contradictory facts that are observed in human pathology.

There are, indeed, cases of septicæmia with fever, and others with central algidity. Occasionally, too, these seemingly opposite conditions may occur in succession in the same individual without the primordial phenomena being at all modified in their appearance.

We may mention, in this connection, what is observed in cases of trau-

[1] Centralblatt, No. 2. 1869.

matic or spontaneous gangrene spreading over a large part of a limb. It has been shown, you know, that when the circulation of the blood has been completely arrested in a gangrenous limb, and when coagula have formed both in the arteries and the veins, the sphacelated portions may become a source of infection. Clinically we are already well acquainted with this fact, but the experiments of Kussmaul have placed this matter in the clearest light. Under the skin of a limb thus apparently separated from the rest of the organism, he injected a certain quantity of iodide of potassium; four hours later he detected traces of iodide in the urine. After this we may no longer doubt but that putrid matters from sphacelated parts can penetrate to the circulatory torrent. The phenomena of infection which they then induce sometimes manifest themselves by intense fever; sometimes, on the other hand, by central algidity. We often notice within this hospital this succession of occurrences in cases of spontaneous gangrene, which usually arise from an atheromatous or thrombic obliteration of the principal arterial trunks of a limb. If, in such cases, the patients resist infection for several days, and especially if the gangrene assume the moist form, the central temperature may progressively fall to 36° or 35° (96.8° or 95° Fahr.); in one case we even saw it as low as 34.5° (94.1° Fahr.). Here death occurred in the midst of symptoms of profound collapse: external algidity, cold sweat, almost imperceptible pulse, etc.

How is it these substances are enabled to lower so rapidly and so considerably the central temperature? It is supposed that they destroy a very large number of blood-corpuscles, or at least suddenly annihilate their respiratory power. In such cases, according to Williams' expression, *necræmia* or death of the blood is produced. Although they preserve their physical character, the globules thus changed have lost their chemical properties. Should this alteration in the blood-disks be general, a rapid fall in temperature ensues.

Still, it is highly probable that, independent of this action, certain substances affect the heart by paralyzing its movements. This happens in the case of bile. Whenever a certain quantity penetrates into the blood, it simultaneously causes the heart to stand still and the temperature to fall (Leyden, Röhrig). Experimentation, to-day, has even gone so far as to tell us what, among the very numerous constituents of the bile, are those which exclusively determine the slowing of the cardiac movements, and what are concerned with central algidity.

Contrary to the ancient idea, we know that all of the bile—or at least the principal fundamental constituents—passes into the blood in simple jaundice, such as that resulting from occlusion of the ductus communis choledochus. Now, in this spontaneous jaundice, as in experimental icterus, the elementary ingredients of the bile are found in the blood and the urine. In both instances, too, a slowed pulse and diminished central temperature may be observed.

Röhrig[1] has proved that these results are due to the presence of the biliary acids; injected *alone* into the circulation, they produce the same effects.

Nothing like it, however, is caused by cholesterin, the coloring, or fatty matters. By the sole fact of diminishing the number and strength of the heart's movements, the biliary acids may induce lowering of the temperature. But Van Dusch and Kuhne have demonstrated that they also possess the power of destroying the blood-corpuscles, and the latter effect un-

[1] Archiv der Heilkunde. 1863.

doubtedly contributes, in great measure, to produce a depression of the thermometric markings.[1]

A remarkable fall in the central temperature likewise occurs in the majority of cases of uræmia.[2]

III.—After what I have just said to you concerning the mechanism by which lowering of the temperature is effected through the influence of the introduction of septic matters into the blood, you will not be astonished to find the same characteristic in *certain organic or functional diseases of the heart*. It is unquestionable that the majority of diseases which weaken the action of the heart tend to produce depression of the central temperature. We can readily understand how flagging of the circulation, carried to a high degree, is a most unfavorable condition for the accomplishment of those chemical acts that maintain the bodily heat. It is also well known that when cardiac enfeeblement is carried to its utmost limits, as in syncope, the blood traverses the capillaries without undergoing any modification therein, and appears in the veins with the bright red color of arterial blood. Now in cases of this kind the central temperature falls, and is even markedly lowered in circumstances where matters are carried less far.

Among the diseases of the heart itself that are accompanied by a diminution of the central temperature, I may mention, as an example, the case of rupture of the organ and extravasation of blood into the pericardium—a case we all saw in this hospital. One morning, in the dormitory, an old woman fell in syncope, and was immediately transferred to the infirmary, where we found her in deep lipothymia, which persisted nearly the entire day. A second syncope occurred just before evening, and death quickly ensued. During the long syncopal period between the two lipothymiæ, the heart was feeble, frequent, and irregular; the pulse almost imperceptible; and the rectal temperature 36° (96.8° Fahr.).

You are conversant with the fact that, in cases of asystolism arising from organic diseases of the heart, there occur from time to time paroxysms characterized by feebleness and irregularity of the cardiac impulse, cyanosis and external algidity. During these attacks we have frequently observed the central temperature fall to 35° or 36° (95° or 96.8° Fahr.), and the paroxysm once over, speedily returns to its normal standard. In *acute pericarditis* and *acute endocarditis*, as we mentioned in our first lectures, we

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[1] Virch. Archiv, xiv.

[2] One of my pupils, Dr. Bourneville, has recently published a series of investigations, whence it results that in uræmia—from whatever cause it springs (parenchymatous nephritis, pyelitis, cystic degeneration of the kidneys, calculous obliteration of the ureters, etc.), and whatever symptomatic form this blood-poisoning assumes (comatose, apoplectic, convulsive)—there is a constant lowering of the central temperature. This fall increases as the nervous symptoms are aggravated, and, in some cases, the *temperature has dropped below* 30° (86° Fahr.). In *puerperal eclampsia*, however, which some authors continue to ascribe to uræmia, Bourneville has observed a constant elevation of the temperature—an elevation that progressively increases from the onset until the fatal termination. 42° (107.6° Fahr.) has sometimes been reached under such circumstances.

Besides, Bourneville has collected a certain number of observations relative to the modifications which the central temperature undergoes in cerebral apoplexy (hemorrhage in, or softening of the brain), which confirm in every respect the results I have published. And further, having taken the hourly temperature in some apoplectic patients, he has demonstrated the influence, upon the thermometric curve, of the production of new hemorrhagic foci, or the effusion of extravasated blood into the ventricles. (See Bourneville: Études Cliniques et Thermométriques sur les Maladies du Système Nerveux. Paris, 1873.)

sometimes notice a rise, sometimes a marked lowering of the central temperature. The same occurs in *peritonitis*. Wunderlich's observations are ample confirmation of the results we have thus far arrived at.

At a cursory view it might appear strange to see a phlegmasia which sometimes occupies a very large extent of surface—the whole of a serous membrane—induce a fall in the central temperature. Such is the case, however: thus, pericarditis is superimposed upon lobar pneumonia; it might be imagined this combination would result in a hyperpyretic ascent of the curve; but this does not occur. The curve either remains stationary, or, in the majority of cases, judging from oft-repeated observations, there is an unusual depression of the tracing. The production of such a depression, during the course of a pneumonia which up to that time has been regular, has often induced us to examine the heart with unusual care, and thus to recognize the existence of a pericarditis which, otherwise, would wholly have escaped us. *Diaphragmatic pleurisy, pneumothorax from perforation, traumatic peritonitis—that arising from perforation* and *internal strangulation*—likewise generally cause, momentarily at least, lowering of the central temperature. True, this is not an invariable result, but it is, nevertheless, quite a common one. Let me add that, in such cases, the central algidity may occasionally persist, or even increase until death, whereas in other instances it may give way to an excessive rise in temperature.

However this may be, we need no longer be astonished to find, if we refer to the results of experimentation, a more or less violent irritation of the serous membranes, the peritoneum in particular, inducing such effects. You know very well that a blow over the epigastric region, the ingestion of cold drinks when the body is covered with perspiration,[1] may bring about sudden death, apparently through the mechanism of syncope. Now, Brown-Séquard has shown that excitation of the semi-lunar ganglia[2] results in the production of syncope. According to him, this excitation of the ganglia is transmitted by the spinal cord to the bulb, is then reflected upon the pneumogastrics, which, irritated in their turn, cause the heart to stop in diastole. When not carried so far as this, the excitation of the great sympathetic plexus may determine *a permanent diminution in the cardiac force*, and hence produce a more or less enduring kind of syncopal (*lipothymic*) condition, along with permanent lowering of the temperature.[3]

It is undoubtedly by an analogous process that great concussion of the nervous system first manifests itself by a syncopal condition with central algidity, accompanied or not by subsequent reaction.

Magendie has proved by experiments, which, Claude Bernard has confirmed,[4] that all severe irritation of the peripheral nerves—such, for example, as the crushing of a limb entails—has the effect of lowering cardiac pressure; the researches of Mentegazza inform us that in such cases the central temperature is diminished.[5]

Following certain traumatic lesions of the cord, Brown-Séquard has ob-

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[1] Consult on this subject the interesting memoir of M. A. Guérard: Sur les Accidents qui peuvent succéder à l'Ingestion des Boissons Froides. Annales d'Hygiéne. Vol. xxxvii. Paris, 1842.

[2] Of the solar plexus.—L. H. H.

[3] See Brown-Séquard: Archives de Médecine. Vol. ii., pp. 440, 484. 1856. Lectures on Physiology and Pathology, p. 159. Bernstein: Herzstillstand durch Sympathicus-Reizung. Centralblatt, No. 52. 1863. *Ibid.*, No. 16. 1864. Eulenberg and Guttman: Patholog. des Sympathicus, in Archiv für Psychiatrie. Bd. ii., Heft. 1. 1869.

[4] Leçons, etc. Vol. i., p. 267.

[5] Schmidt's Jahrb. I., 153. 1867.

served profound collapse, complete suspension of reflex action, and the passage of red blood into the veins. At the same time there is a fall in the central temperature.

IV.—We have just considered the principal circumstances in which the central temperature is lowered. Now, generally, when this thermic depression occurs abruptly, it manifests itself externally by coldness of the surface of the body, and by a train of other *alarming* symptoms. These signs, taken collectively, have been designated under one name, *collapse*, by Thierfelder[1] and Wunderlich, to whom we are indebted for a remarkable study on this clinical point—a point, it must be acknowledged, of which we have been too neglectful.[2]

But collapse may likewise be produced in cases where the central temperature remains normal, or even rises above the normal limits; and according as one or the other of these different forms is exhibited, the prognosis and the therapeutical indications will be found strikingly changed. Collapse is sometimes an almost certain forerunner of a fatal termination; sometimes, again, it undoubtedly indicates a very grave state of affairs, but one that the well-directed resources of the art may, perhaps, carry to a favorable issue. Finally, collapse is at times only an exaggeration of the phenomena which are nearly always observed, to a certain extent, when some varieties of febrile disease terminate with a rapid and favorable defervescence.

From this simple *exposé* it should be immediately apparent to you all how interesting to the clinician the study of collapse must prove, since, every time this group of symptoms is presented to him, there is a problem to be solved, a prognosis to be determined, a peculiar series of therapeutic means to be employed, and, let us add, all these within the barest limits of time, for the symptoms of collapse may only too soon terminate fatally.

These phenomena are very commonly observed in the most diverse diseases of old age, and in those who are weakened by pre-existing maladies or by alcoholismus. But it is when supervening in the course of acute febrile diseases that collapse especially deserves attention. It is under such circumstances that we desire to study it with you, and endeavor to determine its principal characteristics.

Let us suppose a case of lobar pneumonia, and select a well-marked example. Up to the sixth or seventh day all has been in accordance with the established precedent; the pneumonia is very severe, but it has developed regularly; the temperature is 39° or 40° (102.2° or 104 Fahr.), and the external phenomena of the febrile state are well developed. Suddenly, in the space of a few hours, the whole aspect changes; the face is altered, the eyes deeply sunken, the cheeks and nose pale and icy; the extremities are cold and cyanotic; the body is covered with cold sweat; there is great prostration of the forces, and occasionally delirium may set in; the cardiac impulse is feeble and irregular, the sounds seem dull and distant; the pulse is thread-like, now accelerated, now retarded; the respirations are rapid and deep.

What is the signification of this very alarming *ensemble* of symptoms?

The exploration of the central temperature in such a case furnishes us

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[1] Archiv für physiol. Heilkunde, 14te Jahrgang. Heft 2. June 15th.

[2] In an excellent article, Chaleur, in the Nouveau Dictionnaire de Médecine et de Chirurgie Pratiques. (Vol. vi., p. 808. 1867.) Hirtz has brought into prominent notice the interest which attaches to the study of collapse.

most valuable indications: *first*, if, at the very moment when the phenomena of external algidity are being produced, the central temperature is maintained at a high degree, or even rises to a hyperpyretic standard, death is certain. Indeed, dissolution has already begun. Soon it will no longer be a matter of doubt; the pulse will continue to increase in frequency, and laryngo-tracheal râles quickly appear. *Second*, if, on the contrary, at the same time the symptoms of collapse manifest themselves, the central temperature undergoes a marked diminution, or even descends to normal or slightly subnormal limits, the situation of the physician is more difficult, for at times the fatal termination may be already in preparation, and occur without much delay; and again, on the other hand, convalescence may set in at the end of a few hours, and a bad prognosis, hastily given, will thus receive an express contradiction. It behooves us, therefore, to take into most serious consideration the indications offered by the other phenomena which accompany a diminution of the central temperature.

When collapse is but the exaggeration of the ordinary symptoms of a rapid and favorable defervescence—when, at the same time that the central temperature falls, the respiratory movements and arterial pulsations become slower and regular, the prognosis is good, even though some alarming symptom, such as violent delirium, supervene.[1]

When, on the other hand, the central temperature falls, and the frequency of the pulse and respiratory movements continues or increases, the situation is very grave indeed. Death will very soon supervene, despite every effort that may be made. And although we were but recently led to give a favorable prognosis, even when intense delirium was an accompaniment, yet here we must maintain an equally unfavorable one, in spite of the defervescence producing a feeling of comfort in the patient.

Such was the case of a woman, fifty-four years old, enfeebled by uterine carcinoma, who had an attack of lobar pneumonia—a case we had in these very wards. About the seventh day of the pneumonia, at the moment when rapid defervescence occurred, this woman experienced a peculiar sensation of well-being, which has deceived many of you, but which very soon succumbed to the death-struggle.

This is the collapse, which in croupous pneumonia takes place at the period of defervescence as the most usual occurrence. But the same group of symptoms can appear at any stage of the disease.

At the acme of the malady, collapse results in the majority of cases, from some complication, such as pericarditis, violent diarrhœa; or rather, in the aged, it appears from too marked an action of a drug, as tartar emetic or digitalis.

The prognosis you will give varies very much in this class of cases. It depends particularly upon the influence of the means employed to combat the complication, or to repair the damages caused by ill-timed medication.

Again, collapse may manifest itself from the commencement of a pneumonia; in such cases it is usually transitory, soon giving way to a more or less marked reaction; at other times, however, it persists during the whole course of the disease, which then commonly ends most unfavorably.

This *algid* pneumonia is quite rare, even among the excessively debilitated subjects whom we meet with in such great numbers within these walls. Many of you, however, have perhaps observed a remarkable example of this in our wards. The woman, L——, seventy-one years of age,

[1] Weber: Med.-Chirurgical Transactions. Vol. xlviii. 1865.

when attacked with lobar pneumonia, presented from the onset, and during the whole period of the disease, a series of symptoms which made it resemble cholera. The extremities were cold and extensively cyanotic, the face was livid, the eyes deeply sunken, and the voice very faint. There was no diarrhœa. The alvine evacuations were rather scanty ; the urine was pale, diminished in quantity, containing quite a large proportion of albumen. The rectal temperature oscillated between 38° and 38.4° (100.4° and 101.1° Fahr.), never reaching 39° (102.2° Fahr.). The pulse, feeble and almost imperceptible, was between 100 and 108. At the autopsy, the lower and middle lobes of the right lung, throughout their whole extent, presented the most classical characteristics of red and gray granular hepatization ; and the lung itself weighed nearly one thousand grammes more than the left (2 *pounds* 3¼ *ounces avoirdupois*). The kidneys offered no appreciable change.

This variety of collapse constitutes one of the characteristics of the sideral form of the majority of pestilential diseases, contagious and infectious fevers ; thus, we observe it in yellow and typhus fever [1] and in the plague ; it is also met with in paludal (*malarial*) poisoning.[2]

I have often seen it in the small-pox of the aged, where, as you know, it frequently assumes the hemorrhagic form. In such cases, at the very moment when the blackish pustules appear upon various parts of the body, the extremities become cold and cyanotic, the prostration of the forces reaches the utmost limit, and the temperature in the rectum varies between 36° and 37° (96.8° and 98.6° Fahr.).

I am far from having exhausted the subject I purposed to discuss before you, but I fear to weary your attention by multiplying examples ; besides, I have said enough, at least so I hope, to demonstrate to you the exceeding interest which attaches to thermometric studies in the clinic in general, and especially in the case of old age.

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[1] Charcot: Articles Typhus Fever, The Plague, Yellow Fever, in the fourth volume of Élements de Pathologie Médicale. By A. Requin. Paris, 1868.

[2] In algid pernicious fever. Griesinger: Traité des Maladies Infectieuses, p. 64. Paris, 1868.

LECTURE XXII.

SENILE PNEUMONIA.

Summary.—Introduction—Morbid Anatomy—Symptoms—Etiology.

GENTLEMEN:—In our northern climate, diseases of the respiratory organs are the most common and fatal of the diseases of old age.

That we may study this class of diseases intelligently, it is necessary that we should be familiar with some of the more important anatomical and physiological differences in the lungs in adult life and in old age.

The rarefied condition of the lungs in the aged, their increased lightness, the dilatation and rupture of the air-cells, their diminished elasticity, the obliteration of large numbers of their capillary vessels, and the diffusion of carbonaceous matter throughout their substance, tend to modify the pathological changes which occur in the pulmonary diseases of the aged.

The lungs in fleshy old people are of an ashy gray color, studded with black spots and lines. The surface of the pleura is less moist than in adult life. Externally the lungs resemble those of adults; but they crepitate less and have a more elastic feel. The fissures between the lobes change their position, so that in the *left lung* the upper lobe is in front and the lower behind, and in the *right lung* the middle lobe projects downward, the lower one rising behind it, so that it forms a considerable portion of the summit of the lung: thus, pneumonia of the apex may really have its seat in the lower lobe. *Microscopically* the air-cells are about *double* the size of normal adult lung-cells.

In emaciated old people, though the lungs are much the same as in the fleshy, they *cannot be perfectly inflated;* they are bathed in serum; the spots and lines are more distinct, and they crepitate much less.

Again, the lungs of the very aged may present a livid appearance, the apex being larger than the base. Their surface is uneven and looks as if they were "crumpled," and they are surrounded by a large quantity of fluid, which fills the space caused by their wasting; they cannot be fully inflated, and the respiratory murmur is very feeble. They lie close to the vertebral column. Their specific gravity is much less than that of the adult lung, and the lobes are occasionally attached to one another by pedicles. The alveolæ have no definite form, and the cells are very large. The bony thorax accommodates itself to these atrophied, shrivelled lungs.

With advancing years the vital capacity volume decreases with tolerable regularity. The rate of diminution has been estimated to be about one and a half cubic inches each year.

During each hour a healthy adult exhales about one thousand three hundred and forty cubic inches of carbonic acid; now, between the ages of

sixty and eighty the amount, in men, falls to about nine hundred and thirty cubic inches each hour, and in *very* old men it has diminished even to six hundred and seventy.

The same physiological difference exists between the sexes in old age as in other periods of life; thus, women in the sixties exhale only six hundred and seventy cubic inches of carbonic acid an hour, and this continues to decrease in a proportion similar to that in old men.

The precise changes which occur in the interchange of gases in the lungs in old age are not very well understood.

The respiratory activity, the depth, and the force of the acts, all are much less in old age than in middle life; and the predominance of venous blood is a marked feature of old age.

Finally, what the French call "*besoin de respirer*"—want of breath—increases perceptibly as years pass.

With these facts before us, let us study the anatomical changes which take place in the *pneumonia of old age.* In our climate it is the most fatal inflammatory disease of the respiratory organs in the aged. The mortality rate in those over sixty years is about eighty per cent. For this reason, and on account of the very great differences in the disease when it occurs in adult life and in old age, I shall enter into its history somewhat in detail; at least, I wish to make *pneumonia in old age* a subject of special importance in the few lectures which I shall add as a supplement to the interesting and instructive lectures of Professor Charcot on Diseases of Old Age.

Its morbid anatomy may be divided into three stages: a stage of engorgement, of red hepatization, and of gray hepatization. In the *aged* the morbid changes usually begin in the upper lobes, the disease extending downward—the reverse, you remember, of what occurs in adults.

Engorgement.—This stage resembles that of the adult pneumonic lung—the dark red color, the firmness, the pitting on pressure, the doughy feel, and increased weight, all are here. They do not sink in water, the loss of crepitation is far greater than in adult life—indeed, often entirely absent.

On section, a bloody serum exudes, and flows much more freely from the cut than in adult pneumonia. It is now (comparatively) very friable.

This is the only change which you will find in many cases of "sudden death" in pneumonia of the aged.

It is often called congestion of the lungs, and by its gross appearance alone it is difficult to differentiate between this stage of pneumonia and congestion; but a *microscopical* examination reveals, in pneumonia, the air-cells filled with a markedly *viscid* fluid, which, on the addition of alcohol, coagulates into an amorphous, granular substance, containing a number of red blood-globules. The diminution of the lumen of the air-cells by the enlarged capillaries is not well shown in senile pneumonia.

In pulmonary œdema the fluid in the air-cells is simply serum; in pneumonia it is an inflammatory exudation.

In the stage of *red hepatization* the lung is of a deep red color, darker than is usual in adult life, no longer crepitates, and has a distinctly "marbled" appearance, heightened by the dark lines and dots that are present in its normal condition.

The color is sometimes a bright blue or black. The lung sinks in water with a rapidity proportioned to the degree of consolidation, but it rarely falls to the bottom of the vessel, indeed, it frequently remains near

the *surface;* this lightness in the second stage of senile pneumonia is explained by the rarefied condition of the lungs in the aged.

On section, the cut surface may present either a *granular* or a *non-granular* appearance. The granules are much larger than is usual in pneumonia at any other time of life, and, when not irregularly developed, they exhibit a tendency to commingle. Bloody serum exudes from the surface of the cut.

Red *granular* hepatization is very much more common in old age than the non-granular, and the friability is much less than in adults.

A *microscopical* examination shows the large, irregular air-cells to be filled with organized fibrin, red blood-globules, leucocytes, and changed epithelial cells.

In the stage of *gray hepatization* or suppuration, the lungs are still consolidated.

On section, the cut surface presents a marbled drab, or "granite" look, and a copious flow of yellow, opaque pus exudes.

There is more friability than in the second stage, and the lung breaks down on slight pressure, which reduces it to a grayish pulp.

The *microscope* shows the presence of a greater number of leucocytes, and the young cells to have undergone fatty degeneration; the fibrinous plugs having disintegrated, the outlines of the granular elements within the alveoli are indistinct.

These processes may terminate in resolution, gangrene, or abscess.

When *resolution* occurs, the alveolar contents undergo such a fatty or mucoid degeneration that they are readily absorbed.

Pigment, either from the blood or the interlobular connective tissue, often stains the masses, and appears in the expectoration. The lung is now gray-white in color—*never* granular—and a viscid, puriform fluid can be squeezed from the cut surface.

Senile pneumonia terminates in gangrene *much more* frequently than adult pneumonia.

You may find all or part of a lobe of the lung gangrenous. When partial, the gangrene may be limited by an inflammatory zone, or diffused irregularly.

The gangrenous cavities, when near the surface, often cause sloughing of the adjacent pleura, and in nearly all cases the lung-cavity contains a quantity of blackish green, fetid pus. The walls commonly consist of a shreddy material, and vascular bands pass across the cavities. Myriads of bacteria are found in the fluid contents.

The rarefied condition of the lungs in old age seems to favor the development of those small abscesses that are so common after a senile pneumonia. We occasionally find them interspersed throughout the consolidated tissue, several of the alveoli intercommunicating.

Large abscesses may break into a bronchus and establish a vomica, and, if superficial, may lead to pyopneumothorax. An external fistulous opening never occurs in advanced life. Large abscesses, the result of the coalescence of several smaller ones, containing, besides pus, pulmonary *débris,* are rare in old age compared with the disseminated small ones. These abscesses are preceded by intercellular œdema.

In old age, though the right lung is more frequently involved, the difference is far less striking than in adult life. Some observers state that sthenic pneumonia generally occurs on the right, and typhoid on the left side.

Senile pneumonia is invariably accompanied by inflammation of the

minute bronchi, so that it may be difficult to differentiate between a catarrhal and a croupous pneumonia. The pulmonary pleura is much less frequently affected in old age than in adult life.

The lymphatics of the lung are choked with fibrin, red and white blood-globules, and a few endothelial cells; while the deeper lymphatics contain products *identical* with those of the alveoli.

Etiology.—The *predisposing* causes of senile pneumonia are: the age-degenerations, persistent bronchitis, and passive congestion from valvular and other diseases of the heart.

Occupations where sudden changes of temperature occur, and hence, climates where a like condition is present, predispose to its development; all enervating habits, poverty, intemperance, and dyscrasiæ, act as powerfully in old age as in adults. From November until May nearly nine-tenths of the recorded cases of senile pneumonia have occurred.

Bright's disease, pyæmia, and septicæmia are among its predisposing causes; but the last two rarely occur in advanced life.

A recumbent position, long continued, may lead to pulmonary hyperæmia, and thus predispose to pneumonia in the aged more so than in adult life.

Sex is not so markedly prominent as a predisposing cause of senile pneumonia as at other periods of life; indeed, the cases are pretty equally divided between old men and women.

The most frequent *exciting* cause is cold; and dry, sharp cold seems to act, judging from reliable statistics from hospitals for the aged, much more powerfully than moist cold, which rather induces bronchitis.

Fracture of the ribs, pleuritis, and pericarditis excite pneumonia; and it is noteworthy to observe how often, in the aged, such an accident as injury to the hip-joint, for instance, is followed by a pneumonia. It must, I think, be reckoned among the exciting causes, since, even as soon as four hours after the receipt of the injury, the disease is sometimes established.

Lastly, uræmia and certain atmospheric influences, malarial and septic, excite pneumonic inflammation, and "sewer-gas" pneumonia is quite frequently developed in advanced life.

Symptoms.—Senile pneumonia commences very insidiously, and usually runs a *latent* course.

In a few cases it commences with well-marked symptoms in the midst of perfect health, and without any apparent existing cause; or, as in the adult, flying pains in the limbs and chest, occasionally epistaxis, loss of appetite, and a feeling of general *malaise*, may be the precursors of its development.

When intercurrent, senile pneumonia is always latent.

It may begin with a protracted attack of shivering—rarely a distinct chill; when an old person has a distinct chill, pneumonia *almost invariably* follows; for, though less frequent, it is yet more important than in adult life. The same may be said of pain in the side.

According to statistical records from that vast asylum for the aged—The Salpêtrière—it is in March and April that these two symptoms almost always occur, and then the disease assumes the *sthenic* type.

Again, when there is no shivering or pain—which occur in only about half the cases—the onset may be marked by a very slight increase in, or irregularity of, the movements of respiration, a slight elevation of temperature, a feeling of great exhaustion, and a short, hacking cough. In some

cases even all but the feeling of weakness are absent. It may be ushered in by nausea, vomiting, diarrhœa, and collapse.

When an aged person, suffering from chronic bronchitis or asthma, develops a pneumonia, you will very often observe that the cough and difficult breathing which were induced by either of these conditions, undergo remarkable diminution, and the aged patients are scarcely ill; they get up, walk about as usual, perform their meagre daily work, lie down on their beds feeling a "little tired," and suddenly expire. You meet such cases most frequently when chronic cardiac or cerebral disease coexist, although they do not necessarily cause a latent pneumonia.

When not latent, senile pneumonia, once established, progresses with the following symptoms.

The respirations are accelerated; but, as old people rarely complain of dyspnœa, you must count the chest-movements very carefully to recognize the change. There may be a cough, although at times it is so slight as to escape the notice of the physician, nearly always that of the sufferer.

There is pain in the head, usually in the frontal region; and this pain may be followed by a mild delirium, especially toward evening, there being usually more or less disturbance of the intellect, while *very often* stupor or a coma, from which it is momentarily impossible to arouse the patient, comes on and continues throughout the disease.

The face is flushed, sometimes more so on the side of the inflamed lung than on the other.

The pulse is accelerated, the temperature is elevated; and in all cases, on taking the temperature, the thermometer should be inserted in the rectum, for, as Charcot says, in the aged there is very often a marked discrepancy between axillary and rectal thermometry, the latter *only* showing the true heat of internal viscera.

There is great prostration, and the appetite is impaired, while thirst is increased.

I shall now consider the individual symptoms more in detail, and separately.

By far the greater number of pneumonic patients who are advanced in life, *cough;* and the cough, although in the main short, hacking, and painful, yet undergoes greater variations in intensity, frequency, and character, from the fact that in old age pre-existing pulmonary or bronchial affections are so very common.

Expectoration.—In old age expectoration does not appear early, and, even when it does, is liable to sudden suppression. First scanty, gray, and frothy, then yellow or "catarrhal," it finally may become reddish, glutinous, or viscid. The red color is never so marked as in adults; but bloody sputa may accompany acute senile pneumonias whose onset is very severe —these are fatal cases. In the majority of cases the sputa resemble those of bronchitis, the color being opaque, yellowish green. Purely puriform sputa *never* occur in the pneumonia of old age.

Expectoration is always difficult, and is even absent in asthenic, masked forms of the disease; it is rare to see classical variations in the sputa of the aged, such as we witness in those of adult life. Toward the close of the disease the expectoration becomes prune-juice in color; but a chocolate-looking serous sputa may occur soon after the onset, and is a very grave symptom, denoting, as it does, a depraved-blood condition, a "typhoid pneumonia."

A watery, blood-stained, or prune-juice expectoration indicates either a severe and dangerous form of pneumonia, or pulmonary congestion and œdema.

The reason of the non-appearance of the viscid, pathognomonic sputum in pneumonia of advanced life is the rapid transition of the stages—purulent infiltration taking place early.

A microscopical examination of the sputum reveals all the elements described in its pathology as filling the alveoli, and sometimes it affords one of the chief means of its diagnosis.

In asthenic typhoid pneumonia, where the expectoration is slight, it ceases altogether with increasing *prostration*. The sputa contain an exceptionally large amount of sodium chloride.

Finally, the appearance of creamy, abundant sputa on the day of crisis does not hold in old age, since expectoration may be catarrhal from the onset, and throughout its course.

The *rectal temperature* rises to 103° F. or 104° F., sometimes higher, on the first days. Except by the temperature range it is often difficult to determine the exact day of the invasion of senile pneumonia. With daily morning and evening oscillations of a degree or a degree and a half, it continues for three or four days at about the initial point. The rise in temperature does not begin for several hours after the initial chill, if a chill occur.

The temperature may rise progressively, with slight intermittent remissions; or it may suddenly fall, the tracing giving an almost perpendicular line. Both these occurrences are common in senile pneumonia, the first ending in death in the vast majority of cases; while the second, called *the defervescence*, tends sometimes toward recovery, sometimes toward dissolution.

When the onset can be approximately estimated, the temperature will be found to be highest on the third day, unless fatal defervescence occurs.

The *pulse* in the aged is normally more frequent than in adult life. In old men the average normal pulse is from sixty to seventy beats per minute, and in women from sixty-eight to seventy-eight. This is the rule, although in a few instances you may find marked slowness of the pulse-rate in healthy old men and women. Hence, in senile pneumonia the pulse does not afford reliable indications; for the rate, in this disease, might be only fifty, and yet this might be a rapid pulse in this particular instance. When normal, the pulse is from seventy-three to seventy-eight; you rarely find any increase when the pneumonia begins.

On account of the arterial changes in old age, it is best to count the pulse at the heart.

In the pneumonia of adults the pulse is "full," but in old age the pulse has, normally and in disease, a fictitious hardness; when the heart is intermittent the radial pulse may not represent any intermittency or irregularity, and on the other hand it may become feeble and intermittent while the heart is normal and acting with regularity.

The action of cold to the surface in the aged is very quickly indicated by the radial pulse, diminishing its volume and strength; so, if the pulse at the wrist be taken, it should be from the hand that has been under the bed-clothes.

Finally, although the pulse may rise to one hundred and twenty beats or more per minute in senile pneumonia, still it must be regarded as an untrustworthy guide.

The respiration.—In old age the work of the respiratory organs seems

to diminish with that condition of the lungs of which I have previously spoken. The diaphragm is the chief agent in respiration. The scaleni muscles and the sterno-cleido-mastoid becoming nearly useless, you will very frequently notice the head to be thrown back during inspiration, and the whole act of inspiration is imperfect and more or less difficult.

In inspiration in the aged, the enlargement of the chest is chiefly vertical; there is no *lateral* movement whatever. Expiration is sudden and rapid, as a rule; the whole act, however, is of a "panting" character. The number, on the average, of respirations, is twenty-two; and inspiration is to expiration as six to eight or nine.

Very often the inspiratory movements, in old age, are "jerky" and interrupted, the lung becoming expanded only after a succession of efforts. This occurs in perfectly healthy individuals.

There is a marked diminution in the vital capacity of the lungs of the aged.

Again, the altered condition of the mucous membranes of the whole respiratory tract, from imperfect vascular supply, probably interferes with the proper excretion of aqueous and other vapors. For this reason pulmonary œdema is readily set up in senile lungs. The proper amount of excretion does not take place, and hence the fetid breath which is met with in so many old people.

As has been mentioned, old people with pneumonia rarely complain of difficulty in breathing; and you may notice the acceleration in the number of respirations oftener, by palpating the chest than by direct observation, for the respirations may vary from thirty to seventy per minute, and yet no actual dyspnœa exist.

As in the adult, the degree of dyspnœa is directly proportional to the extent and severity of the pneumonic process, except *in pneumonia of the apex*, when there is always intense dyspnœa, so much so that severe dyspnœa in the aged should always direct you to a physical exploration of that part of the chest.

"Catchy" breathing—a mere exaggeration of what is physiological in old age—is one of the most frequent forms of abnormal respiration, in the pneumonia of advanced life.

When dyspnœa is very intense, and the pneumonia is at the base of the lung, it tells of nervous exhaustion, and is very serious.

As you will remember, the general causes of dyspnœa in pneumonia are the diminution of the breathing surface; the influence of the fever; the pain, which is exacerbated by movements; the enfeebled heart-power; and the greater demand for oxygen, on account of the rapid tissue-metamorphosis.

Pain.—Even in pleuro-pneumonia of the aged, pain is never very intense; it is rather an uneasy, dull feeling, occupying no particular spot, and very frequently is diffused over the whole chest.

Tenderness on palpation or percussion is not so marked as in the adult.

Pain, if present in senile pneumonia, is referred to various localities: the pit of the stomach, the region of the xiphoid cartilage, the nipple, the loins, the hypochondrium, or even the side opposite to that which is the seat of the pneumonic process; but it is always anterior. In pneumonia at *the apex* it is transitory or completely absent.

In "typhoid" pneumonia there is no pain; but, as the disease progresses, a sense of "oppression" occurs, which increases with the increasing weakness.

One side of *the face*, more than the other, has a "mahogany" colored flush upon it, not diffused as in typhus fever, but as a circumscribed spot. This flush is often the first objective sign of senile pneumonia. The heat of the skin is greatest in the morning. Three or four days from the onset of the pneumonia, herpetic eruptions appear upon the cheeks, lips, and nose.

If the lips become blue, it denotes vaso-motor disturbance, and is a very grave sign. As the disease advances, the face loses this dusky hue and becomes sallow—a very dangerous symptom—and the surface-heat gives place to a cold, clammy perspiration.

At my next lecture I shall continue the consideration of the objective symptoms and physical signs of senile pneumonia.

LECTURE XXIII.

SENILE PNEUMONIA—*Continued.*

Summary.—Symptoms—Physical Signs—Differential Diagnosis—Prognosis—Treatment.

GENTLEMEN:—*Headache* usually lasts throughout an attack of senile pneumonia, and is apt to be accompanied by delirium of a mild type, when *the apex* is the seat of the inflammatory process. In asthenic, typhoid, or "nervous" pneumonia, there is low, muttering delirium, and a constant desire to get out of bed. These symptoms, however, do not of themselves indicate any serious cerebral disturbance.

Of the symptoms referable to *the alimentary tract*, anorexia is the most constant; and, while patients do not express a desire for drink, they yet take with avidity the fluids which are put to their lips.

The tongue at first is whitish; later on it becomes dry, shrivelled, and coated with a thick, brown fur, and is protruded with difficulty. The teeth, gums, and inside of the cheeks are likewise covered with thick, brown sordes.

The stools are dry, and, as a rule, the bowels constipated.

Some think that the *liver* undergoes passive hyperæmia during a pneumonia, the mechanism being the same as in certain cardiac derangements; and thus they account for the jaundice which so frequently accompanies senile pneumonia. But it is more in accordance with to-day's pathology to regard the jaundice as of hematogenous rather than of hepatogenous origin; caused, in other words, by a change in the blood itself, the direct result of the pneumonic process.

Jaundice is more frequent in cases where the pneumonia is located at the apex.

The *urine* is scanty, dark red or brown in color, and of high specific gravity. Bile-pigments are found in it. There is an increase in urea, and, on cooling, the urine becomes turbid from precipitation of the urates. The most characteristic change is the great diminution of chloride of sodium.

Albumen may be present, and the higher the temperature, the more albumen may we expect to find, other things being equal.

The signs which indicate the mode of termination of senile pneumonia vary.

If a case is tending toward a *fatal termination*, the face becomes sallow; the skin cold and clammy; the alæ nasi and extrinsic muscles of respiration are called into play; the heart becomes feeble, rapid, irregular and intermitting; the temperature either rises rapidly or falls quickly and continuously; the inspirations are gasping, there are great apathy and somnolence, and gradually coma comes to usher in the fatal issue. You may sometimes hear, at a distance from the patient, the râles which result from that œdema of the lungs which comes from heart-failure.

When *resolution* occurs in senile pneumonia, it is generally by crisis. The temperature falls suddenly, sometimes below normal; there is a return of the appetite, a general feeling of returning comfort, and if there has been prune-juice sputa, the red color disappears and a catarrhal expectoration takes its place. This, however, is not a very frequent termination of senile pneumonia.

Convalescence is slow, months elapsing before complete recovery is reached.

If the pneumonia terminates in *gangrene*, typhoid symptoms appear very early. The face is generally pale, sometimes of a deathly hue; the eyes are sunken, the breath is fetid, and the whole body emits a cadaverous odor.

The sputum, if present, is of a blackish green color, of a putrid odor, and contains shreds of decomposed and decomposing lung-substance. If pulmonary gangrene is a sequel of pneumonia, death occurs with symptoms of the profoundest collapse, usually within five days from the time of the initial chill.

The formation of *abscesses* in senile pneumonia is never evidenced by any well-marked symptoms. There may be rigors and hectic; the expectoration may become purulent, copious, and of a grayish tint, sometimes containing fibres of pulmonary tissue, with the physical evidences of a lung-cavity.

Finally, before studying the physical signs of senile pneumonia, there remains a not unimportant variety which is called *bilious pneumonia*—an unfortunate name, since the term bilious is too often, at the present day, applied to any group of symptoms that seem to have some connection with liver derangement. Diarrhœa and vomiting are present in this form, and occur very early. The vomiting is "bilious" in character, and icterus is generally more or less strongly marked. Somnolence and stupor likewise may occur in this form, which is an extremely unfavorable symptom.

Another variety, which is peculiar to old age, is remittent pneumonia. All the symptoms, after energetic treatment, cease for a time—to return, however, with greater severity the next day; and as this lull in the disease seems most promising, a prognosis might be given which would suffer embarrassing contradiction in the future developments of the case.

Physical signs.—Before the physical signs of senile pneumonia can be accurately estimated—if, indeed, this is possible, the physiological modifications of old age must be carefully considered.

Respiration, as has been said, is mainly diaphragmatic in old age; the average number per minute is from twenty-one to twenty-two, and inspiration is to expiration as six to nine.

The physical signs are modified by the bony union of the different parts of the sternum and ribs, by curvature of the spine that is generally well marked in old age, by the rigidity of the bronchial tubes, by the rounded form of the chest in advanced life, and, finally and most important of all, by that rarefaction of the lungs to which reference has been made.

Percussion.—From the above-mentioned considerations, one would expect, what actually occurs—a very clear percussion-note, and occasionally one that, compared with an adult chest, is abnormally resonant.

When the air-cells have ruptured to any considerable extent, you may elicit, on firm percussion, a resonance as well marked as occurs in slight adult emphysema (the reverse of what occurs in middle life); the clavicular region near the median line gives a dull percussion-sound on account of the great arching of the sternum and the great deposition of carbonaceous material at the apex of the lungs.

The infra-clavicular and mammary regions are extra-resonant.

The lungs being pushed back to the spinal column, the sternal region is less resonant, and may often sound almost *dull;* the heart-limits can be accurately marked out, on account of its being uncovered by lung-tissue.

The *scapular* and *supra-scapular* spaces are less resonant than in the adult, on account of the tilting of the scapulæ, due to curvature of the spine.

Auscultation.—There is a loss in the vesicular element of the respiratory murmur in old age. The respiratory sound resembles closely that produced by a rather forceful expulsion of air from the compressed lips, and is diffuse in character, sounding as if the incoming column of air met a thinned and readily vibrating membrane; the murmur is louder and less soft than in adult life.

In lungs where the septa are torn and the alveoli greatly distended and irregular, the respiratory sound is bronchial in character.

The intensity of the respiratory murmur in old age is variable: at one moment it is loud, at another, hardly perceptible; and it varies not only thus in the same individual, but also in different individuals of the same age and condition of body.

The voice-sounds are very loud; occasionally there is even bronchophony, and in very extensively atrophied lungs this bronchophony has a vibrating or catching character, causing it to resemble very closely œgophony.

Finally, it is an almost *physiological* condition for old people to have a bronchorrhœa; hence, mucous râles may be constantly present during the whole period of advanced life.

To the physical signs of pneumonia one always turns for confirmation of his diagnosis, but here one will be misled who has in mind only the usual adult signs.

First Stage.—*Inspection* and *palpation* in the first stage of senile pneumonia furnish little positive information.

Percussion may reveal slight dulness; this is very rarely the case until the lung has reached the stage of red hepatization, and even then it may be so slight in character as to leave grave doubts of its existence.

Auscultation.—Very early in the disease the respiratory murmur is feeble and indistinct over the affected portion, while the lung that is not involved assumes for the time all the characters of a normal adult respiratory murmur.

Again, the breathing over the pneumonic spot may be intensely *puerile,* and in such a case will usually be rough, interrupted, or broken in character.

Tubular breathing may sometimes be heard at the root of the lung, and then the respirations are generally markedly feeble.

A peculiar bronchial *souffle* is often heard over the inflamed lung, and in some cases this is audible in the superior sternal region.

As soon as the pneumonic exudation occurs in the adult, the distinctive crepitant râle is heard, but in advanced life a distinct crepitant râle in the first stage of pneumonia is rarely present.

But subcrepitant râles, and large, moist râles, resembling to some extent what we hear in capillary bronchitis, but oftener analogous to the râles of a simple bronchitis, are heard during the period of exudation.

The reason for the almost invariable absence of the crepitant râle is, it seems to me, to be found in the physiological condition in old age; the alveoli are dilated, their walls are thinned and frequently perforated, and the lungs themselves are retracted from the chest-wall.

Sometimes, when you make the patient cough violently for a few minutes, and then have him take a *deep* inspiration, you may hear fine crepitation; but, upon closer examination, it will still be found to be subcrepitant in character, in no wise differing from that of capillary bronchitis.

Soon the crepitation, whatever character it may assume, extends over the chest, from the general bronchitis that so soon accompanies the disease, and masks, in whole or part, the respiratory sounds.

It may be stated, as a general rule, that the feebler and more superficial the respirations, the less distinct will be any adventitious sounds.

The dilation and rigidity of the bronchi that are physiological in old age, of course favor the development of *bronchial breathing*, which, indeed, is oftentimes the first physical sign of the pneumonia. One of its peculiarities, when occurring in the stage of engorgement, is that it is *limited to the root of the inflamed lung.*

Hence, this is an important sign, and one that should be always listened for in all doubtful cases. In the unaffected lung the breathing is often puerile, and sometimes accompanied by dry, transitory bronchial râles; and in a few exceptional cases, when the lower lobes are involved, the upper give evidences of hyperæmia.

Adjacent to the inflamed portion the respiratory murmur is very much weakened.

Second Stage.—*Inspection* in some cases reveals diminution in the expansibility of the affected side, but this is not constant.

Palpation may or may not show increase in the vocal fremitus.

Percussion.—What is *dull* in old age might be regarded clear in the adult; hence, when dulness on percussion is said to mark the stage of consolidation, the term is only relatively true. Still, when superficial, there may be actual as well as relative dulness. The dulness is best marked posteriorly.

Auscultation.—Tubular or bronchial breathing marks the second stage of senile pneumonia, and the sounds are even more intense than in adult life; small gurgles or mucous râles generally persist throughout this stage. Bronchophony is not very well marked, even when present; while ægophony, from the quavering voice in advanced life, is far from infrequent.

Third Stage.—*Inspection* gives negative results.

Palpation may or may not reveal increase in the vocal fremitus, according as it was present or absent in the previous stage; but in general vocal fremitus decreases.

Percussion shows the dulness to be decreasing—the chest again becoming very resonant.

Auscultation shows the crepitating sounds to be louder; and the gurgles loud and large, often heard at a distance from the pneumonia.

The *râles redux*, then, are not, as such, distinctive of, or peculiar to the third stage of senile pneumonia. The sound heard at this period of the disease is called a muco-crepitating sound, by which is meant a form of subcrepitant râle produced in tubes intermediate between the bronchioles and the larger bronchi.

Finally, as has been hinted, the physical signs of pulmonary *abscess* in the aged are very generally wanting; distinctly localized gurgling and cavernous respiration *may*, when taken in connection with the rational signs, suffice for an approximate diagnosis, but the great rarity of abscess in old age should make us cautious in its diagnosis. The sputa will greatly aid us in such a case.

The physical signs of senile pneumonia are subject to greater variations than ever occur in pneumonia in the adult; and often they do not even follow the course, irregular as it is, that I have just described.

Gray hepatization, or abscess, may be reached without any distinctive auscultatory signs being heard, even on repeated and careful examination.

Even in "resolution" the *râle redux* may be absent; dulness and bronchial breathing being *immediately* followed by normal (senile) resonance, without crepitation. This occurs most frequently in the "typhoid" variety.

Differential diagnosis.—The question very naturally arises: How can senile pneumonia be diagnosed if it may run its course without either expectoration or physical signs, the symptoms on which we so implicitly rely for a diagnosis of pneumonia in adult life?

Grisolle says that an exploration of the thoracic organs in pneumonia of the aged gives negative results in a majority of cases, and that we must base our opinion, 1st, on the extreme frequency of pneumonia in old age; 2d, that, of all the phlegmasiæ of advanced life, pneumonia is the one which is oftenest *latent;* 3d, that, of all acute diseases in old age, pneumonia produces the highest range of temperature and the greatest prostration.

So, when an old person has a chill, followed by febrile movement and great prostration, and when the superficial and splanchnic portions of the economy (other than thoracic) do not account for it, *pneumonia* may be diagnosed, even though all its (adult) diagnostic signs are absent.

A few practical hints as to the means of diagnosis seem to have their place here.

The characteristic expectoration *may* be obtained on violent coughing after several swallows of fluid have been taken.

On causing the patient to cough and expire violently, dyspnœa, crepitation, and tubular breathing may be present for a short time.

When there is no cough, repeated and *deep* inspirations may develop it and dyspnœa also.

Senile pneumonia may be confounded with *capillary bronchitis* and *pleurisy.*

In both capillary bronchitis and senile pneumonia there is a muco-crepitant or subcrepitant râle; but in pneumonia it is circumscribed to a spot three or four inches in diameter, while in capillary bronchitis it is diffused, and heard best over the posterior portion of the base of *both* lungs.

When both lungs are affected, the attacks are consecutive in pneumonia, while they are simultaneous in capillary bronchitis.

There is dulness, or at least a diminution of the resonance in pneumonia, whereas an exaggerated percussion-note is present in capillary bronchitis.

In pneumonia there may be bronchial breathing, while in capillary bronchitis the vesicular murmur is feeble—often *absent.*

In pneumonia there is but *one,* the initiatory chill, while in capillary bronchitis there are several slight attacks of chilliness during its course. The *pulse* in capillary bronchitis may run up to 140 or 150 in the early days, while 120 is about the limit of the pneumonic pulse-rate.

The temperature is *always* higher in senile pneumonia than in capillary bronchitis. Cyanosis is a marked and early symptom of capillary bronchitis, while it occurs only late in pneumonia, and generally is a forerunner of death.

Finally, a microscopic examination of the sputa—when they occur—will

decide as to the condition of the alveoli, and whether pneumonia is or is not present.

A *pleurisy* with effusion sufficiently extensive to cause dulness would cause bulging of the intercostal spaces, with more or less displacement of the heart or liver, whereas these never occur in pneumonia.

Again, the percussion-note would be flat over a large extent of surface in pleurisy, while a pneumonic spot would be evidenced by *localized* dulness over a small extent of surface. Besides, the line of flatness changes with the position of the patient in pleurisy, and is, of course, always stationary in pneumonia.

There is usually *absence of vocal fremitus* in pleurisy, while in pneumonia it may be increased, or at least normal.

The respirations are "catching" in pleurisy, and panting in pneumonia.

If bronchial breathing exist with a pleurisy, it is not as sharp, defined, and tubular in character as in pneumonia, but rather subdued and diffused. Increased voice-sounds, bronchophony and œgophony are present in pneumonia, while the voice-sounds are feeble over the pleurisy.

A chill, when it introduces a pleurisy, is very slight, often absent; a patient with pneumonia generally gives a history of a distinct, often a severe chill.

The temperature in pleurisy is two, three, or more degrees lower than in pneumonia.

The face is flushed in pneumonia, while in pleurisy it is pale and anxious.

The pain in pleurisy is sharp and stitch-like in character, located at the nipple of the affected side, while in pneumonia it is often absent, and when present is dull, diffused, and referred to various remote regions, all of which have been mentioned.

It may be added here that the diagnosis of a pleuro-pneumonia is made upon a combination of the chief signs of senile pleurisy and pneumonia.

Prognosis.—This is always grave, and the greater the age of the patient the less are the probabilities of recovery.

Statistics do not give pneumonia its proper place among fatal diseases of old age. I believe it to be the most fatal of all the acute diseases at this period of life, for the number of autopsies that have been made in cases of "sudden death," in individuals of advanced years who have had "low" fever, "nervous" fevers, etc., exhibit in the vast majority of cases red or gray hepatization (of greater or lesser extent) that was not suspected during life. Many of the most reliable modern authorities—those who have had the largest experience in the hospital practice of the aged—state that nearly nine-tenths of those over sixty-five die of pneumonia.

When pneumonia is single and confined to the lower lobes, the prognosis is much better than when it has its seat at the apices of the lungs.

The prognosis is also more favorable when delirium is absent and the pulse is not irregular or intermittent. When the pulse-rate does not exceed 110 or 120, and when the temperature-range is not above 103°, the prognosis is favorable. Thick, creamy sputa are very favorable signs.

Prune-juice expectoration, the presence of any complication, extreme dyspnœa, excessive prostration, a sallow face, all these are very unfavorable signs.

It is difficult to establish the average *duration*, but, in general, a primary adynamic pneumonia lasts from six to ten days; in fatal cases the *seventh* day is rarely passed.

The first stage rapidly passes into the second, perhaps in four or six hours.

The stage of purulent infiltration, if it occurs, is reached very frequently before the third day, and in some instances within thirty-six or forty-eight hours from the onset.

Pneumonia may be complicated by pulmonary œdema. Bright's or heart disease may have pre-existed, and thus act as complications. Death results from *cardiac failure*, from asthenia, or from complications.

Treatment.—The therapeutics of pneumonia in advanced life is very different from that of adult life: often the third stage is reached before the aged patient consults you.

Never bleed in senile pneumonia.

In the Salpêtrière it seems that an emetic, when not especially contra-indicated, was usually given in the first stage of pneumonia.

Tartar emetic is never permissible in old-age pneumonia.

The physicians of the Montpelier General Hospital regard ipecacuanha as an heroic remedy in senile pneumonia.

English physicians regard the nitrate of potash as efficacious; the Germans prefer hydrochlorate of ammonia.

Drastic purgatives and blisters should never be employed in the treatment of senile pneumonia.

The most important thing to be accomplished is to sustain the heart; antipyretics are rarely necessary.

Alcoholic stimulants and concentrated fluid nutrition are our main reliances in the treatment of this affection. In fact, absolute rest and the judicious use of stimulants are all that are required in the majority of cases.

Iron and quinine may be employed in moderate doses as aids in the sustaining plan of treatment.

In typhoid, asthenic pneumonia, large quantities of quinine may be administered with benefit; and the diarrhœa occurring with typhoid pneumonia must be promptly checked by vegetable astringents.

LECTURE XXIV.

SENILE CHRONIC CATARRH OF THE BRONCHI.

Summary.—Morbid Anatomy—Etiology—Symptoms—Differential Diagnosis—Prognosis—Treatment.

GENTLEMEN:—Chronic bronchial catarrh is a very constant attendant of old age. It sometimes receives the name of "the old man's winter catarrh," one of its chief characteristics being its tendency to recurrence, the attacks increasing in severity and duration with each return of cold weather, until the individual is rarely free from it.

It may be chronic from the onset, coming on late in life; or it may be the sequela of acute attacks occurring in adult life or early manhood.

Morbid anatomy.—Senile bronchial catarrh may have its seat in any portion of the bronchial tubes; thus, it may be limited to the larger bronchi, or it may extend into the capillary tubes. Its anatomical changes are the same, whether it has its seat in the larger or smaller tubes.

Usually the inflamed membrane has a gray or slaty blue color.

The changes begin, primarily, in the mucous tissue, and subsequently involve the entire bronchial wall.

Numerous small elevations and depressions are to be found upon it, often giving it a velvety look and feel.

Occasionally it is of a deep brownish red color, and has a well-marked whitish look throughout the whole extent of the tract. Not infrequently the membrane is atrophied instead of hypertrophied, and it is then very thin, presenting neither fibrous nor cartilaginous tissue; but, in either case the vessels are dilated—often tortuous. Vegetations, appearing as small, papillary elevations, may also occur.

The bronchial glands are usually more or less enlarged, their mouths standing wide open and gaping. Two colored zones surround the gland, one around the centre, the other around the base. The process results in weakening of the walls, so that the violent and prolonged fits of coughing to which the individual is subjected cause dilatation at certain points. This dilatation, or *bronchiectasis*, is occasionally uniform—oftener, however, there is sacculation. The sacs, or ampullæ, may attain the size of a hen's egg, and, when several are agglomerated, they resemble greatly a tuberculous excavation. The surrounding tissue always shows the characteristics of a *peri-bronchitis*, all the changes being best marked at the periphery of the lung. Other portions of the tubes become narrowed, and so alternate stenosis and dilatation are very common conditions.

The membrane may be covered with a thick, viscid layer of yellow, puriform material, or again (*catarrh sec*), a semi-transparent, glairy, mucoid matter may cover, in a thin layer, the affected portions—the microscope revealing numerous pus-cells and columnar epithelium.

The submucous coat exhibits an increase in connective tissue, and when

this increase involves the pulmonary parenchyma, we may have induration of the lung as the result. Just here it may be mentioned that the process resulting in fibrous induration may extend from without inward, *i.e.*, from the pleura; but it generally results from the inhalation of irritants, giving rise to a condition called "stone-cutter's phthisis."

Accumulations of cheesy matter are occasionally found at points scattered irregularly throughout the bronchial tract, oftenest back of those localities where stenosis is marked.

Again, degeneration, or decomposition of the secretions may result in *ulceration*, and thus a focus for gangrene of the lung is sometimes established. The ulcers are usually at the posterior portion of the tubes, and rarely extend deeper than the cellular tissue which connects the mucous membrane to the parts beneath. When, however, they are deeper, a fistulous opening may occur into the œsophagus, aorta, pleural cavity, large blood-vessels, the pulmonary parenchyma, or even *externally*. Large or small abscesses may be excited by the perforation of a bronchial ulcer into the lung-substance, or else inflammation may be set up as a result.

You very frequently find emphysema developed as the result of a long-continued bronchitis, although under such circumstances a hereditary taint will rarely be absent; and from what has been said concerning the physiological (senile) changes, it is easy to understand why emphysema is the most common pulmonary pathological attendant of chronic bronchitis.

That ulceration which we so often meet with in chronic laryngeal disease—the diffuse and follicular—*may* occur in the bronchi, although it is one of the rarest accompaniments of bronchial catarrh.

The cartilaginous structures of the air-passages sometimes suffer ossification. In very old subjects the ultimate ramifications of the bronchi may become changed into osseous spiculæ, having minute canals—the cavities of the bronchi—running through them. In old subjects, too, the bronchial cartilages may become so brittle as to break when handled; or, during life they may extend into the cavity of the tube, break off, and be expectorated. Tubercles are not infrequently found irregularly disseminated throughout the lung; such cases are more numerous the more advanced the years.

Etiology.—Age is a most certain predisposing cause of chronic bronchial catarrh.

Vice of constitution is also a powerful factor in its causation, and first, under this head, stands gout, then rheumatism, and chronic skin diseases, such as chronic eczema and psoriasis. When the skin diseases disappear, the bronchitis makes its appearance.

Syphilis, chronic Bright's disease, and chronic alcoholismus likewise predispose to this affection.

Exposure to sudden changes of temperature—and thus a changeable climate, bad hygiene, overcrowding, poor ventilation, and certain unknown atmospheric conditions, act both as predisposing and exciting causes to bronchial catarrh.

Organic diseases, such as reside in the heart especially, act in a purely mechanical and easily comprehended manner, first causing hyperæmia of the bronchial mucous membrane, and subsequently a state of chronic catarrh.

Finally, it is to be remembered that certain old persons exhibit a strong tendency to bronchial catarrh, even when the more marked predisposing and exciting causes are absent.

Symptoms.—In the declining period of life, nothing is more common

than to find, during the winter months, a cough and expectoration which the patient avers he has had "on and off" for years. It may not materially interfere either with his general health, but it is nevertheless "troublesome," generally abating or entirely disappearing in the spring and summer months.

A moist, cold atmosphere or unusual changes in diet aggravate it, and sudden exposure is very certain to bring about what may be called an acute attack, or what the individual calls a "spell." Going upstairs painfully increases the shortness of breath, and there is a general feeling of tightness or fulness across the chest.

There are no febrile symptoms; but more or less dyspnœa and wheezing are constantly present, the shortness of breath oftentimes simulating an asthmatic attack.

The simplest form, the one in which the larger tubes alone are involved, is where a moderately severe fit of coughing, usually occurring in the morning, is followed or attended by the expectoration of a yellow, yellow-green, or grayish mucus, which soon after becomes white.

In some cases it is glairy in character, and in nearly all a microscopic examination shows it to be made up of pus-cells and mucus, with, perhaps, a *few* epithelial cells.

The expectoration is oftenest nummular, although it occasionally runs together, and when very viscid is of an irregular, ragged, round shape, floating in a turbid, gray mucus.

The name *pituitous catarrh* has been applied to chronic bronchitis when the sputum resembles the white of an egg mixed with water, and is ropy, clear, and gelatinous; again, when with considerable coughing only a ball of pearl-gray, semi-transparent mucus is expectorated, it has been denominated "*dry*" catarrh—of course a misnomer.

In this form the sputa are generally tasteless or slightly saline, or of a sickly sweetness and without odor.

In the enfeebled and intemperate there is a form of chronic bronchitis where there is loss of flesh, and where night-sweats occasionally occur. This is marked by *violent* fits of coughing, followed by the expectoration of a gray, viscid, tenacious muco-purulent mass, which sometimes is watery, but which soon regains its muco-purulent character.

The odor is sweetish, as a rule, but may often be slightly fetid; it is never odorless.

The amount varies; but the average quantity is about a pint a day. During one of the paroxysms of coughing the effort to dislodge the mucus is often so strenuous that vomiting sets in, and the contents of the bronchi and stomach are ejected simultaneously.

The expectoration contains, besides pus and mucus, epithelial cells in comparative abundance, albuminous matter, and now and then small portions of bronchial tissue.

In this class of cases there is *slight* dilatation of the tubes and a small amount of attendant induration, but to a less extent than occurs in what will later be described as *bronchorrhœa*.

Chronic bronchitis frequently occurs with gout, spasmodic asthma, and emphysema. (In the latter case the emphysema is the primary pathological condition.)

In these cases the cough is violent, *prolonged*, and paroxysmal. For a time it seems as though the patient could not regain his breath; his face becomes red, then livid; the veins swell; the eyes are suffused; the head seems as if bursting, so that the patient clasps it in both hands; the mouth

is opened in a sort of gasp; the jugulars are swollen; and vomiting is often the result of the diaphragmatic pressure; finally, a small, rounded pearl of tenacious mucus is expectorated, the patient takes a deep breath, and though immensely relieved, is weakened by the "fit," and is exhausted for some time after.

Bronchorrhœa, also called "pituitous flux" and "bronchial flux," is usually associated with heart disease, and is marked by violent fits of coughing—less intense, however, than in the last-named variety; and the expectoration, *often a pint at a time*, of a glairy, colorless, transparent mucus, with froth at the top, looks precisely like the white of an egg in water.

Two or three pounds may be discharged in two hours; sometimes large quantities are, as the patients say, vomited up of a morning, so easy is, at times, the ejection of the fluid. These, however, are exceptional cases.

This form, frequently accompanying cardiac disease, is very apt to cause grave symptoms when the patient is exposed to sudden changes in temperature; or, especially, to strong winds; and it is this form which is liable to merge into, or be accompanied by, pulmonary œdema.

Extreme fetor of the sputa is generally associated with slight febrile excitement; and it may be stated as a general rule, to which, however, there are many exceptions, that fetid expectoration is associated with bronchiectasis.

The sputa are *abundant*, gray in color, and, in rare instances, moulded in the form of the smaller tubes.

Occasionally there is a slight blood-tinge in the expectoration, which indicates the existence of superficial ulceration; sometimes the expectoration becomes brownish and *fluid*, the microscope showing it to contain pus-globules mingled with fatty granules, crystals of *cholesterin* and *margarin*.

Mixed with water, the pus readily dissolves, thus showing that but little mucin is present; and the solid parts of the sputa sink to the bottom of the containing vessel, there to form a greenish sediment, often with whitish, cheesy plugs intermingled.

Physical signs.—Inspection shows more or less labored respiration, always aggravated by exercise, motion, or excitement. Sometimes the chest presents a more concave shape than normal.

Palpation may reveal an increase in the vocal fremitus when thickening and dilatation of the bronchial tubes exist. Such increase in the vocal fremitus is local. If dilatation of the air-tubes exists, the vocal fremitus will be diminished.

Percussion.—There is a loss in the pulmonary resonance in all well-marked cases.

When emphysema exists with bronchitis (and this is usually the case), the percussion-note will be somewhat tympanitic in character.

Auscultation.—Feeble respiration is a constant attendant of chronic bronchitis in the aged. The variety of adventitious sounds heard during respiration is, perhaps, greater than in any other pulmonary disease.

The respiration is harsh in character, often accompanied by a vesicular quality, but not *tubular;* it also has a peculiar, sonorous element.

When the tubes are narrowed, and when stenosis is marked, high-pitched, hissing, and sibilant râles are heard, confined generally to the finer bronchi.

A sonorous, cooing sound has its seat in the larger tubes, where also are produced large gurgling sounds that result from the bursting of large bubbles.

Similar conditions in the smaller bronchial tubes produce sibilant and subcrepitant râles.

Since the bronchi of both lungs are affected at the same time, all varieties of dry and moist râles can be heard at different portions of the chest; a sonorous is very near to a sibilant râle, and not far off is a subcrepitant or muco-crepitating râle.

After a copious expectoration, the moist râles may wholly disappear for a time, and the auscultatory sounds vary greatly at different examinations.

The sonorous and sibilant râles often completely mask the finer sounds. Fine sounds, like the musical notes from miniature organ-pipes, palpable to the patient himself, are frequently heard.

When *emphysema* coexists, the expirations are prolonged and of a low pitch, and dyspnœa is much more marked.

Bronchiectasis is evidenced at times by well-marked gurgling, the gurgles being of large size, and most abundant at the lower portion of the lung.

Finally, in long standing chronic bronchitis there will usually be some displacement of the heart downward, on account of the accompanying emphysema.

Differential diagnosis.—Senile bronchitis is very often mistaken for senile phthisis.

In phthisis there will be—when occurring with such symptoms as are common to chronic bronchitis—more or less retraction of the chest-walls; while in bronchitis the chest is more frequently convex or bulging.

Percussion in phthisis gives well-marked loss of resonance at the apex of the chest, which never occurs in bronchitis.

Auscultation in phthisis may reveal a cog-wheeled or jerky respiratory murmur, a prolonged *high-pitched* expiratory sound, or the respiratory murmur may be completely tubular in character.

These signs are not present in chronic bronchitis; and *if* the expiration is prolonged, it is of *low* pitch.

In advanced phthisis the dulness becomes wooden over the affected lung, there is amphoric or "cracked-pot" resonance on percussion, and on auscultation, cavernous or amphoric breathing with the metallic tinkle may be heard.

These signs of a cavity are sometimes present in bronchiectasis, when it will be impossible to make a differential diagnosis; in fact, such a form of chronic bronchitis may be regarded as bronchial phthisis.

In all doubtful cases the history of the disease is important, and where the physical signs leave you in doubt, the presence or absence of fever, the character of the expectoration, and the amount of emaciation will decide the question.

Prognosis.—Next to pneumonia, bronchitis is the most fatal disease of old age—some place it at the head of the list, but reasons have been given (see Etiology of Pneumonia) for questioning this statement.

The duration of senile chronic bronchitis varies very greatly; there are many cases where the old man's winter-cough has existed for twenty years. It may be relieved, but not cured; the elements that oppose or favor recovery are the following.

The older the patient, the longer he has had the disease, the more feeble his condition, the more marked the predisposition to bronchial affections, the less are his chances of permanent relief or of partial recovery.

Again, a fetid expectoration, which is constant or increasing in amount, is an unfavorable sign; as are also evidences of bronchiectasis or emphysema.

Gout predisposes to acute attacks and gives permanency to the changes in the bronchial tubes.

Emphysema and *bronchiectasis* predispose to pneumonia and pulmonary œdema.

Hepatic congestion, abdominal dropsy and general anasarca are frequent attendants of chronic bronchitis; seventy-five per cent. of such cases are complicated by a small, granular kidney.

Intestinal catarrh is often a complication of chronic bronchitis, the chief signs being pain at the scrobiculus cordis, pain in the stomach, a sense of constant gnawing, a capricious appetite, indigestion, and flatulence. The liver is almost always simultaneously engorged in these cases.

Death, in senile bronchitis, may result from pneumonia, gangrene, or œdema of the lungs, or from the renal, and rarely from the gastro-intestinal complications.

Treatment.—A careful study of each individual case is important for the successful treatment of chronic bronchitis; indiscriminate routine treatment, or the administration of favorite prescriptions, may do much more harm than good.

A warm, dry climate, at a moderately high altitude, should be selected as a residence for this class of patients; they should remain indoors as much as possible when the prevailing winds are east. Night air and cold are to be avoided. The sleeping-room should be well ventilated, and the temperature should be kept between 65° and 70° Fahr.

Flannel should at all times be worn next the skin, and the cutaneous functions should be regularly stimulated by Turkish baths, rubbing, and moderate exercise.

The diet must be simple and highly nutritious, for only in cases where the nutritive functions are active can any permanent relief be hoped for. The bowels must not be allowed to become constipated, and alcoholic stimulation must be regulated by the indications of each case.

The most important thing to be accomplished in its *direct* treatment is the *removal of its cause.*

When a weak heart is evidently its predisposing cause, digitalis may be administered. When *gout* is prominent, colchicum, the iodide of potassium, and alkalies often produce marvellously beneficial effects. Steam inhalations of hyoscyamus, conium, or stramonium are usually of great service in cases of gouty bronchitis.

When *syphilis* is made out as the chief factor, iodide of potassium and the compound calomel pill will be found useful in connection with the iodine-spray inhalations.

When there is anæmia, which is very apt to be present in aged females, cod-liver oil, iron, and moderate exercise in the open air, will be found of the greatest service; while moderate stimulation, combined with quinine and strychnia, will sometimes give marked relief.

When chronic *skin diseases* alternate with a bronchitis, arsenic and sulphate of zinc are to be administered for a long period.

In emphysematous patients, iodide of potash, dilute nitric acid, and the ethereal extract of the acetate of iron, must be given daily for a long period.

The rule is that a general tonic plan should be followed in all cases.

In bronchorrhœa, and when the amount of secretion is excessive, vapor

inhalations of naphtha, creosote, balsams, copaiba, or ammonium chloride often have the effect of checking or diminishing it.

When a severe attack of bronchorrhœa comes on, a hot-air or vapor bath may be given in connection with sedatives or stimulants, according to indications. Dry cupping may be occasionally employed in such cases with benefit.

When there is evidently a lack of power to expectorate, ammoniacal preparations, squills, senega, and the resins frequently afford prompt relief. When a *large* accumulation takes place in the bronchi, do not hesitate to administer an emetic of sulphate of zinc.

When the cough is so constant and harassing that it interferes with sleep, opium and cannabis indica may be given for relief.

Local treatment consists in the application of dry cups, sinapisms, blisters, or stimulating liniments to the chest. Croton-oil liniment is most frequently used. Any of these measures may be employed, when they have been found to afford relief. Bloodletting is *always* contraindicated.

Hepatic and gastric complications are best relieved by an occasional mercurial purge.

LECTURE XXV.

ASTHMA.

Summary.—Nature—Etiology—Symptoms—Differential Diagnosis—Prognosis—Treatment.

GENTLEMEN:—Asthma stands next to bronchitis in the list of troublesome affections of the respiratory apparatus in old age.

It is now regarded as a disease of purely nervous origin. It has no special morbid anatomy.

Tonic muscular spasm, or contraction of the circular muscular fibres of the bronchial tubes, is the essential element of an asthmatic paroxysm; and their consequent narrowing, as evidenced by unmistakable physical signs, is a mechanical result. An analogue of this is found in spasmodic stricture after a gonorrhœa; for a bronchitis is known to be the exciting cause in many cases of asthma. The bronchial membrane is first vascular, then occur certain catarrhal processes with which you are acquainted, and subsequently, through reflex nervous irritation, there is spasm of the bronchi, which is *asthma.*

Etiology.—The causes may be divided, for convenience, into primary and secondary; meaning thereby that one class affects *directly* the air-tubes, the other some organ or part remote from the respiratory apparatus.

When the *asthmatic tendency* is present, there are a multitude of articles which cause its development, such as smoke, dust, a fog, emanations from stables, hay, roses, burning sulphur matches, sealing-wax, the odor of horses, cats, and other animals—any of these may be sufficient to bring on a paroxysm of asthma.

In some a neighborhood where filth prevails is one where their asthma is best, in others the approach to such a place brings on an attack.

The same is true of dry air and moist air, of high lands and low lands, the mountain and the seashore. Some asthmatics cannot sleep or be free from asthma except in certain parts of the house; and, again, the native place of some patients is the only spot where they enjoy immunity from the disease, while others can never be free from it at home. These idiosyncrasies are strongest in advanced life, and are by no means imaginary; for, to witness the suffering of certain old persons when they disregard their well-known individual idiosyncracies, will be enough to show how real all these causes are, and at the same time show how powerful *nervous* influences are in producing asthmatic paroxysms.

Asthmatic attacks may be excited by the ingestion of certain articles of food; this is the "*peptic*" asthma of some authors. The peripheral pneumogastric filaments in the stomach's wall probably suffer irritation in these cases, and thus secondarily induce spasm in the parts supplied by the other ramifications of this nerve.

Bronchial catarrh is probably the most frequent of all the secondary causes of asthma in the aged. The tendency to asthma is often latent until a general bronchial catarrh is developed. Heart disease, "*cardiac*" asthma is the least common of all. It is produced in the vast majority of cases in a purely mechanical manner: by causing a passive hyperæmia of the bronchial mucous membrane.

The retrocession of gout and rheumatism, the abrupt disappearance of chronic eruptions upon the skin, the stoppage of an habitual discharge—any of these may be followed by a paroxysm of asthma, and it may be from the latter cause that the drying up of old ulcers is capable of producing the malady. Sometimes aged asthmatics have applied at the hospital for irritating dressings to keep open old ulcers, so certain were they of the effects of their healing.

Among *comparatively* rare causes are cold feet, a loaded rectum, an enlarged prostate gland, and organic diseases of the brain.

Finally, there is sometimes a well-marked hereditary predisposition, and, again, *no cause* can be discovered to satisfactorily account for the asthmatic paroxysms.

Symptoms.—The symptoms of asthma may be divided into those which are present during the attack and those of the interval.

Prodromal symptoms are of rare occurrence in the aged; but when they are present they consist of languor, drowsiness, and depression, or the opposite condition of extreme lightness of spirits. There is very frequently a large quantity of "hysterical" urine passed some hours before the attack; indeed, all the prodromata of asthma are eminently *nervous* in character.

The attack proper—whether premonitory symptoms have or have not preceded it—usually begins with a sense of constriction across the chest, and the peculiar paroxysmal dyspnœa. When, as is common in old age, dyspnœa is habitual, there is a sense of suffocation, and the patient begins to wheeze, cough, and perhaps has a fit of sneezing attended by running at the nose.

There are anorexia and flatulent distention of the abdomen, with wheezing and increasing dyspnœa; these may continue for a day or more.

Again, there are no warning symptoms at all, and the attack comes on suddenly and unexpectedly.

Early morning—from two to four o'clock—is the usual time for an asthmatic to be waked out of sleep by difficulties of breathing. Dyspnœa increases and becomes suddenly or gradually intense, so much so that it seems sometimes as if the patient could not live; every muscle is called into play that aids in enlarging the chest, and all possible positions are assumed.

The aged patient rushes to and opens the window to get air.

The shoulders are elevated and brought forward.

The face is pale or dusky, the features are altered, the mouth is wide open, the nostrils dilated, the veins in the neck and forehead are turgid, and the sufferer feels as if death were impending.

Though he is in a dripping perspiration, the extremities—the lower especially—are cold; the pulse is small, rapid, and thready.

If expectoration have existed, it ceases in the acme of the paroxysm, and not until this is resumed will the fit end, *not because* the voiding of the collected mucus relieves the paroxysm, but because, when the bronchial spasm passes off, *expectoration* follows.

Even when the attack begins "dry" there is always expectoration at the end, the *result* (not the cause) of the spasm.

True asthmatic sputa consist of grayish pearls of mucus, like tapioca, and are usually free from either pus or watery material.

So intense may be the paroxysm that streaks of blood appear in the sputa; and in some (rare) cases, this amounts to actual hemorrhage.

There is no rule as to the duration of one of these paroxysms. They sometimes continue for half an hour. I have known a paroxysm of asthma to continue for seventy-two hours in an old man of eighty.

Less violent attacks may continue without remission for five or six days; again, each attack is made up of several shorter ones, the intensity of which varies greatly in different cases.

The longer the attacks the slower and less abrupt the departure, and the more profuse the sputa.

After an attack the patient is often reduced in strength and exhausted, has aching limbs, depression of spirits, and a feeling of "soreness" in all the respiratory muscles.

In "cardiac" asthma, the symptoms are very much aggravated, and the patient being usually conscious of the heart-lesion, has not even hope left in the midst of his sufferings, and often prays for death to come and end them; after an attack of this kind, convalescence is very slow.

The *character of* different attacks in the same individual varies. If at first they are violent and exhausting, later they may assume a milder form, and lose somewhat their periodical character and run into each other, so that the individual is never entirely free from paroxysmal dyspnœa.

There is always at first a periodicity to asthmatic attacks, the intervals between the paroxysms varying in length.

As a rule, the longer the individual has been subject to asthma the shorter the interval between the attacks; this is especially true in the very old.

It may be mentioned here that no physical signs are present, except during the paroxysm.

Some have stated that the urine exhibits a remarkable diminution in chloride of sodium and urea immediately after an attack, while later both resume their normal standard.

Usually, after an asthmatic attack has passed, most asthmatics are comparatively well; the patient feels certain he will be free, for a certain time at least, from another, and meanwhile can enjoy good health.

Physical signs.—During the paroxysms *inspection* shows labored breathing, but no increase in the number of the respirations. Inspiration is shortened, while expiration, though effected by abrupt and violent efforts, is longer than normal. The muscles of the neck are immovable and the vessels are distended.

Palpation gives normal vocal fremitus.

Percussion elicits slightly exaggerated resonance.

It is *auscultation* that enables us, if doubt could exist in well-marked asthma, to obtain distinctive results. The inspiratory murmur is heard very faintly or is wholly absent; this is especially the case in very old subjects. No vesicular quality is present, and the expiration-sound is prolonged and low-pitched.

Sibilant and sonorous râles, high-pitched, hissing, wheezing and squeaking in character, are heard over the whole chest, and especially over and between the scapulæ in the very old.

Often these dry sounds have a musical character. When moist sounds

are heard, it is either because chronic bronchitis complicates the asthma, or, as the paroxysm is drawing to its close, mucus collects in the tubes. Asthmatic sounds are oftentimes heard at some distance away from the sufferer.

The number and kind of sounds heard during the beginning of a severe asthmatic attack cannot be enumerated; they are constantly changing their locality, and also their nature in the same locality.

In some cases—the tubes perhaps not being all affected, or equally so—the respiratory murmur is heard in patches; it is apt to be exaggerated in these spots.

All the sounds are loudest during expiration, and in some instances they may even be limited to that period. During the interval between the paroxysms there are no abnormal sounds.

Differential Diagnosis.—Asthma may be confounded with laryngeal spasm, angina pectoris, pulmonary œdema, bronchitis, emphysema, and pericarditis with effusion.

I.—Spasm of the glottis is attended with voice-changes never present in asthma; and, besides, there are the physical signs of asthma.

II.—*Angina pectoris* is attended by pain of a lancinating, cutting, or stabbing character, while asthma is, as its name indicates, a gasping for breath. Angina pectoris is accompanied by *no* physical signs of pulmonary abnormalities, while the patient himself usually attributes everything to the heart.

III.—*In pulmonary œdema* there is dulness on percussion over the œdema, while in asthma the chest is extra-resonant.

The râles in œdema are liquid bubbling râles, while asthmatic sounds are *dry*.

Pulmonary œdema is early accompanied by profuse watery expectoration, while *after* an asthmatic fit small pearls of gray mucus may be expectorated.

IV.—*Bronchitis* is of slower advent than asthma, and is accompanied by the *distinctive moist râles*, while asthma is marked by many and various *dry* sounds, and is very sudden in its advent.

The respirations in bronchitis are always more rapid than in asthma. The character of the sputa will also aid in the diagnosis, for it is usually purulent in the former, and *never* so in asthma.

V.—*Emphysema* is never wholly unaccompanied by dyspnœa, while asthmatic patients do enjoy intervals of immunity.

Emphysema is accompanied by its characteristic percussion-note, vesiculo-tympanitic in character, and this is never present in uncomplicated asthma.

Prolonged low-pitched expirations exist, usually in emphysema; and in asthma, the duration, if prolonged, is *never* low-pitched.

Finally, the aspect of the patient, the barrel-chest, and the change in the position of the heart which accompanies emphysema, will enable you to make a diagnosis.

VI.—Perhaps one of the diseases most likely to be mistaken for asthma when it occurs in old age, is latent pericarditis with effusion. It may be, and often is, accompanied by dyspnœa, which is spasmodic in character, and pain may be wholly absent; hence the mistake in the diagnosis.

The chief signs by which pericarditis is to be recognized, are feebleness and often irregularity of the pulse, faintness, diminished cardiac impulse, obscure heart-sounds and increase in the præcordial dulness; and, negatively, in pericarditis there will be no râles, no history of previous asthmatic attack—indeed, no pulmonary symptoms whatever.

Prognosis.—Senile asthmatics are generally long-lived; a paroxysm of asthma rarely terminates fatally, and when death occurs during an attack, it is most generally from pulmonary œdema or congestion.

It is the attendant circumstances that have to do with the establishing our prognosis; and also the fact that severe and prolonged attacks may bring on emphysema of the lungs and right cardiac hypertrophy, and dilatation incident to obstruction in the pulmonary capillaries during these paroxysms.

When *organic* diseases exist, as for example, a chronic bronchitis, then the disease must be looked upon as incurable; and the more advanced the age, the more unfavorable is the prognosis; hence, in this class of patients the disease may be put down as *incurable.*

When no abnormal lung- or heart-sounds are heard during the intervals between the attacks, the outlook is better than when the reverse is the case.

Asthma may be complicated by chronic bronchitis, emphysema, bronchiectasis, pulmonary œdema and congestion, cardiac dilatation and hypertrophy.

Death, if it occurs, usually results from the pulmonary complications.

Treatment.—In the treatment of asthma, two things are to be considered: how we may relieve the paroxysms, and how we may prevent their recurrence. In other words, the treatment is to be palliative rather than curative.

When a patient has a paroxysm of asthma, the first thing to be done is to remove the exciting cause when it can be reached. When the stomach has been overloaded, an emetic should be given; when the rectum is overfilled, an enema should be administered; when smoke, dust, or the emanation from anything which induces the fit, these should be removed, or the patient withdrawn immediately from their influence. It is the same with localities and emotions, or nervous impressions.

The removal of the cause may alone bring about speedy relief. But when this is not the case, or when no cause is discoverable, then certain remedial measures may be employed.

The room in which this class of invalids spend most of their time should be large and well-ventilated, the curtains should be removed, and all obstruction to the ingress of sunlight and fresh, cool air avoided. The idiosyncrasies of old people suffering *this neurosis* should be respected.

The next step is to select those remedies best adapted to the case, and the choice depends much upon the individual's experience and individual constitutional idiosyncrasies; most asthmatic patients know what is best suited to their case.

The remedies may be divided into three classes: *depressants, sedatives,* and *stimulants.*

1. *Depressants.*—The chief drugs in this class are antimony, ipecacuanha, lobelia, and tobacco. The object in the use of these is to completely relax the spasm, and whichever drug is chosen it should be exhibited until the required effect is produced.

Ipecacuanha may be given to produce nausea, and tobacco should be smoked until nausea and a feeling of faintness come on, when the attack of asthma ceases. Mild tobacco should be used by old people and vomiting ought never to be induced by its use.

2. *Sedatives* act either locally on the pulmonary nervous apparatus, or *generally* upon the whole nervous system.

Those of the greatest value in asthma are chloroform, opium, stramonium, ether, cannabis indica, hyoscyamus, and the fumes of nitre-paper.

There is, perhaps, no agent that so promptly relieves as chloroform, and this, too, when all others have failed. Great care should be exercised in giving chloroform to the aged, and the condition of the heart must always be carefully considered. The relief is in any case but temporary.

Ether is pleasanter than chloroform, both to inhale and in its results: a combination of the two will in some cases be found more effective than either alone.

Hypodermics of morphia, when combined with atropine, act well in many cases; this combination acts much more powerfully than either drug *alone*.

Nitre-paper, one of the oldest and best known remedies for asthma, is prepared by dipping filter- or blotting-paper in a saturated solution of saltpetre. The fumes of the nitre-paper give no relief when bronchitis accompanies the asthma, and as these two maladies coexist so often in old age, it is seldom of much service in senile asthma.

When used, the room must be filled with the fumes from the burning paper. Its action is not well understood.

Stramonium leaves, when smoked, will in some bring about speedy relief, while in others no relief follows their use. Internally, in the form of *extract*, stramonium does not afford the same relief as when the dried leaves are smoked. The *datura tatula* is regarded by some as more efficacious than *datura stramonium*. Many old asthmatics will not go to bed until they have "had their stramonium pipe."

It may be added here that the habitual use—in either sex—of tobacco seems to have a favorable influence, and in some cases gives complete relief.

Conium, hyoscyamus, and belladonna sometimes afford relief, but much less certainly than the stramonium. They may be tried, however, in obstinate cases.

The *nitrite of amyl* has not proved as successful as its physiological properties would warrant us to expect.

3. *Stimulants.*—The two chief *stimulants* to be employed in the treatment of asthma are coffee and alcohol; coffee is the most efficient, and should be taken upon the occurrence of the first symptoms of an asthmatic attack. It must be very strong and without milk. Two or three cups are sometimes necessary to ward off an attack. It should never be administered on a full stomach, for then it *may* excite or aid in inducing an attack.

Alcohol is sometimes of great service in the treatment of asthma. It is of little importance what form of alcohol is employed, provided it is hot and strong; in other words, it must be given as a "hot toddy." It must be given in sufficiently large doses to produce its intoxicating effects. As it is sometimes the only remedy that will relieve, and as larger doses are required at each attack, there is danger that its use may become habitual.

Asthmatic patients who are relieved by alcohol, as a general rule, have their paroxysms occurring during sleep, and as during sleep the nervous system is more depressed than during waking hours, these individuals are more susceptible to the exciting causes of the asthmatic fit when asleep than when awake.

As in all nervous conditions the remedy which is first "the specific," so called, will later become useless, and then a new one must be tried, and so the changes may be rung on half a dozen drugs with success.

Compressed air has not been found serviceable in the treatment of asthmatic attacks, and the inhalation of oxygen does not exercise any markedly beneficial influence.

The treatment in the interval is essentially hygienic, although some state that, in cases whose cause is undiscoverable, the iodide of potassium not only prevents or delays the attacks, but even prevents their return. Again, when cutaneous disorders appear to irregularly alternate with asthma, the internal use of arsenic will be found of service.

Quinine, five or ten grains a day, will sometimes prevent asthmatic paroxysms, and when old asthmatics suspend it for even a short time, the paroxysms may return with greater severity.

One, if not the most important duty of the physician in treating senile asthma, is the regulation of the diet of the patient.

Breakfast, at eight o'clock, should consist of half a pint of green tea, or of coffee, and a little cream and two ounces of yesterday's bread. Dinner, at one o'clock, two ounces of fresh beef or mutton (no fat), and the same quantity of well-boiled rice or stale bread; and in about three hours after dinner half a pint of weak brandy and water, and as much toast-water as desired.

No water is to be taken within one hour before dinner or supper, or until three hours after.

Flannel should constantly be worn next to the skin, and the flesh-brush may always be employed with benefit.

Moderate exercise only is allowable, and cold bathing of the chest must be prescribed with caution in old people. Remember that old asthmatics are always prone to cold feet; hence, woollen socks are to be worn, and all chilling of the body carefully avoided.

When tonics are indicated, iron and quinine will be found best for asthmatics of advanced life; as to climate, experience is here the only and the best guide.

It may be said that all confirmed asthmatics are dyspeptic. It is therefore important to study the idiosyncrasies of each individual case, in the interval between the paroxysms.

LECTURE XXVI.

ATHEROMA.—FATTY HEART.

Summary.—Atheroma—Morbid Anatomy.
Fatty Heart—Varieties—Morbid Anatomy—Etiology—Symptoms—Differential Diagnosis—Prognosis—Treatment.

GENTLEMEN:—Before we commence the study of the diseases of the heart and blood-vessels in old age, I desire briefly to call your attention to the more important modifications which the heart and circulatory system undergo in advanced life.

The *heart* of the aged differs in many respects from that of adult life. Contrary to what occurs in the other organs, it does not diminish in size or weight as age advances; in very many cases it undergoes actual hypertrophy. Indeed, it may be said to be physiological for the heart to exceed by one-twelfth its normal adult weight.

Its average weight in males over sixty is eleven ounces—in females of the same age, nine and a half ounces. It is *broader* than in adult life, and the cardiac substance, in the majority of cases, is pigmented.

Yet in emphysema, and in those very old people who seem "to shrivel up," the heart may be no larger than a base-ball, and somewhat resemble it in shape.

The increased size and weight of the heart are due to progressive thickening of its walls; the capacity of both the auricles and ventricles are increased; and there is an enlargement of the auriculo-ventricular openings.

The ratio of increase in the capacity of the right and left cavities is equal; but the left ventricular walls increase in *thickness* much more than the right. This thickness of the left ventricle varies considerably with the place measured, since its thickness rapidly diminishes from base to apex; at the latter point the endocardium and the tunica propria almost meet, so thin is the ventricular wall.

The auriculo-ventricular orifices undergo a uniform enlargement; but the aortic orifice enlarges much more rapidly and to a greater extent than the pulmonary, and some state that aortic insufficiency is physiological in old age.

The valves are thickened, and so is the endocardium, either in points or throughout its entire extent.

The ascending portion of the arch of the aorta is generally dilated to a considerable degree; this is undoubtedly the result of the blood-current striking upon the aortic walls, whose elasticity is gradually diminishing. It may thus *attain its double calibre*, and the walls generally become much thicker in such cases. This thickening takes place in *all* the tunics of the artery.

The loss in the elasticity of the aorta is due to connective-tissue increase and to fatty or to calcareous degeneration.

Again, in many old subjects there is minute injection of the aorta and pulmonary artery just as they leave the heart. The reflected portion of the pericardium is then quite lax, as is also the subcellular tissue; and the arteries and veins therein are enlarged and tortuous.

The *veins* in old age lose much of their elasticity, and increase in size and in the thickness of their walls; all *venous* changes are most marked in the lower half of the body.

The amount of blood is diminished in old age (*senile anæmia*), and nearly all the viscera are in an anæmic condition. It contains less solid constituents, especially red globules and albumen. It is clearer, more fluid, contains more cholesterin, and coagulates much more readily than in adult life, which accounts for the coolness of the extremities in old age; at least this is one of its causes.

At this point it is important that I should say a few words concerning *atheroma*, for this is the great factor in the production of all senile pathological changes.

Atheroma.—The term *atheroma* includes a pathological condition of the arteries which may be, and often is, a combination of chronic arteritis, fatty and calcareous degeneration.

The earliest stages of atheroma are marked by an infiltration of cells into and beneath the inner coat of the vessel. These cells are leucocytes mingled with connective-tissue cells. This hyperplasia causes bulging of the tunica intima toward the axis of the tube. At first the swelling is soft, and can be easily removed. Retrograde metamorphosis gradually occurs as a result of defective nutrition, the new cells undergo fatty degeneration, and a soft, yellow mass, called an "*atheromatous focus*," results; or, the lining membrane rupturing, the pultaceous contents are swept into the circulatory torrent, and we then have the *atheromatous ulcer*.

Again, when neither of these processes occurs, the liquid portion of the mass becomes absorbed, cholesterin is formed, and then in the deeper layers there is a débris of broken-down cells, cholesterin crystals, fat-granules, and some of the original fibrillated tissue, which, subsequently calcifying, *becomes a calcareous plate*.

A frequent change in old age consists in *fibroid* thickening of the inner coats of the vessel.

In the aorta, just above the aortic valves, you will see, in old age, these white or yellow-white patches extending over a considerable space.

These calcareous plates are friable, transparent, and *inelastic*, sometimes thickened at their irregular rims, and when the vessel is dilated in spots, and the plates are numerous, we have what has been called "*senile arteritis deformans*."

In *old* age the arteries of the brain and heart are affected in an almost equal degree, as well as those of the chief glands.

The same processes and the same results occur in old age in the valves of the heart, which are sometimes markedly cribriform and corrugated.

In connection with *atheroma* may be described the *white spots on the heart and pericardium*, which are unimportant, except that they are *very* common in advanced life, and are not found at other periods.

They are usually on the anterior surface of the right ventricle, and vary in size from that of a pin's head to that of a two-cent piece. They are very irregular in shape, and sometimes can be dissected off as a false membrane. There are two theories concerning them—an inflammatory and a non-inflammatory; the former is the better received and supported theory.

They occasionally occur on the pericardium, and are far more common in old men than women.

The almost cartilaginous consistence which the vessels have, when atheroma is of long standing, may lead to sufficient retardation, or arrest, of the blood-current to cause arterial coagula; and then gangrene of the extremities, fatty degeneration of the heart, or cerebral ramollissement may follow. This is sometimes called the "marasmic thrombosis," and there is, undoubtedly, a diminution in the influence exerted by the vaso-motor nerves upon the arteries. From this impediment may also come dilatation and tortuosity of the *veins*.

In individuals in advanced life, it is rare not to find a chronic peri-arteritis accompanying the lesions of the heart and vessels which I have just described. And where the aortic arch gives origin to the great vascular trunks of the head and the upper extremity, rings of bone-like matter very often surround the orifice of the branch.

Having considered the usual condition of the heart, vessels, and blood-mass in old age, and the lesions which constitute senile *atheroma*—that lesion which so often is followed by so many and terrible results—we are prepared to consider the modifications that occur in the heart and pulse in *normal* old age.

Percussion.—The normal area of dulness in the adult, which, you remember, is of a triangular shape, with the base immediately below the junction of the left third rib with the sternum, and the apex on a line of the cartilage of the sixth rib, is usually increased about half an inch in *all directions* in old age.

The apex-beat is lower down, being, as a rule, three instead of two inches beneath the nipple, and is carried nearer the axillary line than in adult life.

When the organ is overlapped by an adherent portion of the lung, or when there is emphysema, percussion may wholly fail to accurately map out the size of the heart, and to discover its exact dimensions.

Auscultation.—As a rule, the heart-sounds may be said to be *duller*. The first sound, especially, seems in old age as if it were prolonged and muffled.

A moderate aortic insufficiency is usually unaccompanied by any marked auscultatory symptom, but may give the same murmur and the same area of diffusion as in adult life.

Pulsations in the veins.—It is stated by many writers that the tricuspid valves are normally in the aged slightly insufficient, and this is hence regarded as a sort of safety-valve. Now, it has been said that the auriculo-ventricular orifices become larger in old age, and thus jugular pulsation, from both these causes combined, is a quite frequent occurrence in old age. Now and then they are markedly distended, and you may notice a thrill.

When aged subjects are excited, it is frequently the case that the veins above the clavicle present a marked undulatory movement, and this may be perceptible in the emaciated, even beyond the region of the neck.

The pulse.—The heart's action in old age, although no less powerful than in adult life, is nevertheless accompanied by an impulse which is less visible. The number of pulsations in aged females is always greater than in males. As examples of extreme slowness of the pulse may be mentioned the cases where the pulse in a man eighty-eight years old was twenty-nine, in a woman eighty-two, thirty-six; and in a man eighty-nine, twenty. That such slow pulses are compatible with normal *senile* cardiac conditions, there can be no doubt.

In old age, when no lesions are present, it is not uncommon for the

heart to present irregularities both in the strength of its beats and in its rhythm. These two conditions may both occur in the same individual, or there may be only a loss of rhythm.

It is a peculiar and interesting circumstance that when acute diseases occur in old people whose hearts are thus irregular, the rhythm and strength become, during the disease, perfectly normal, to change again when recovery occurs. There is no satisfactory explanation of this occurrence.

The pulse in advanced life is quite characteristic: it is, hard, wiry, and deficient in elasticity, so that its unvarying rigidity makes it an uncertain means of investigation.

The radial artery shows the changes of age better than any other: as you slide the readily moving skin over the artery, the calcareous plates often give a sensation similar to that of passing the finger over a tube filled with shot.

Failure to appreciate these arterial changes has undoubtedly led to many fatal bloodlettings in old age, for the older the subject the firmer and harder seems the arterial impulse.

As I have already stated, it should always be the rule to count the pulse at the heart in old age.

These are the more important changes in the heart and vessels in the aged. By keeping them constantly in mind, much of error may be avoided. The first cardiac disease that claims our attention is fatty heart.

Fatty Heart.

Fatty degeneration is the commonest degeneration in old age; it is one of the prominent factors in what is denominated senile decay.

Fatty degeneration may be either partial or general, and, as in adults, presents itself under two forms.

"Quain's fatty heart" is a degeneration of the primitive muscular fibres, and is properly so denominated; while the deposit of fat in the areolar tissue of the heart, or the replacing of the connective tissue by fat, is in reality, *fatty infiltration.*

Morbid anatomy.—In advanced life, this degeneration is most often met with in the left ventricle near its apex; next in order comes the right ventricle, and lastly, the right auricle and left auricle.

The first change in this degenerative process is a loss of the nuclei of the primitive muscular fibres, with a simultaneous loss of their striated appearance. Granules, completely filling the sarcolemma, make their appearance, and from having the look of albuminoid matter, change to fat-granules and oil-globules, rarely larger than a red blood-corpuscle; they are generally arranged in very nearly even rows, and at last absolutely fill the sarcolemma.

The normal size of the fibres remains unaltered and all the fibres are not affected to the same extent.

Some have divided this degeneration into two forms: the granular and the fatty. But the former is more usually regarded as only a stage of the latter.

A heart which is the seat of this change is of a paler color than normal, more opaque, and its tissue is friable, breaking down with a soft, granular fracture; there is a distinct loss of cohesion. The organ is usually normal in size (that is, the normal size for advanced life); it may be hypertrophied or dilated, the latter being the much more frequent condition. In some

very old people the discolored and flabby heart is noticeably diminished in size.

When the degenerative process is partial, the heart will collapse when removed from the body, and exhibit a mottled appearance, the spots varying in color from a drab, gray, or dirty brown hue.

These opaque patches are unevenly distributed over the heart, and occur in the papillary muscles, the columnæ carneæ, and especially in the fibres just under the endocardium. They are readily broken by pressure of the finger, and under the microscope will show large oil-globules and fat-granules, many of which have escaped into the adjacent tissue. On section the blade of the knife is covered with the fatty material, and the organ may leave oil-stains on paper. This *partial* softening is so common at this period of life, that some French writers call it *senile softening*.

The coronary arteries may be obliterated, calcified, atheromatous, or normal; there being no *necessary* relation between abnormalities of these vessels and fatty degeneration of the heart, except when it is secondary to muscular hypertrophy.

In *fatty infiltration* there is merely an increase of fat in the areolar tissue. Striæ of fat may be seen lying among the muscles, or so thickly is the heart encased in it, that it seems to be but a mass of fat. This accumulation is always most marked at the external surface of the organ, diminishing toward the endocardium. It *may* establish true fatty degeneration, or fatty metamorphosis, by its pressure, and thus an *intra*-stitial may become an *inter*-stitial process.

This is sometimes called senile obesity of the heart; and you will usually find in the hearts of old people depositions which are quite extensive around the base of the ventricles, in the line of junction between them and the auricles, and at the origin of the large vessels.

Along the course of the coronary vessels the fat is contained in oval or round cells whose average diameter is about one one-hundredth of an inch. This differs markedly from true fatty metamorphosis.

Etiology.—Fatty heart is essentially a disease of old age, although it may occur at any age. The liability to it steadily increases with advancing years.

Fatty infiltration, overgrowth, or senile obesity occurs usually in those who have an excessive development of adipose elsewhere. It is twice as frequent in the male as in the female, and the influence of heredity cannot be doubted; there is, in a large number of old people, a constitutional tendency either inherited or acquired. Sedentary habits exert a marked influence in developing, or aiding the development of fatty infiltration of the heart.

Fatty heart often comes on not only with *senile* marasmus, but also with that marasmus accompanying Bright's disease, gout, phthisis, cancer, and chronic alcoholismus. It is worthy of remark that fatty heart from such causes seldom reaches a point where it seriously interferes with its action.

In old age a very frequent cause of cardiac degeneration is interference with the coronary vessels. Such interference may be due to atheroma and calcification of these vessels, to embolic obstruction, external compression, from pericardial thickening, or impaired recoil of the aorta from any cause whatever. Poisoning from phosphorus or phosphoric acid, and perhaps disease of the cardiac ganglia may lead to this diseased condition of the heart.

Finally, in some instances, the causes of true fatty degeneration or metamorphosis of the heart must be regarded as yet undetermined.

Symptoms.—The objective signs of fatty degeneration of the heart in old

age are obscure. Indeed, some authors (Balfour among them) state that it cannot be recognized during life with certainty. Moderate fatty degeneration will go unrecognized, and sudden death may occur when there is no suspicion of its existence during life. The disease progresses slowly and gradually, and the symptoms vary in proportion to the extent of the process.

The aged patient is incapable of attending to any business that requires the slightest physical exertion; the muscular power is steadily diminished.

The breathing is easily embarrassed, and fits of dyspnœa and palpitation are easily induced, and during the paroxysms there may be acute pain and faintness, the attacks simulating those of angina pectoris, while tinnitus aurium, giddiness, and other phenomena of a deranged cerebral circulation, are very common characteristics.

During the paroxysm the liver enlarges and the respiration becomes feeble and irregular, frequently sighing in character.

Attacks of syncope, the result of cerebral anæmia, increase in frequency and severity as the disease progresses, and it is a peculiarity of fatty heart that the patient himself is aware, on a retrospective view, that there has been a steady and decided loss of muscular power.

Independent of all "anginal" attacks, you will now and then meet cases where pain is seated over the cardiac or sternal regions, or shoots down the arm during the greater part of the time; it then becomes one of the most troublesome symptoms.

The countenance is sallow, "pasty," or pale; perhaps livid from venous stagnation; the extremities are cold, and, in a few cases, œdematous. The tissues of the body are flabby and there are the usual evidences of arterial degeneration; *arcus senilis* is a valuable sign of this form of cardiac degeneration, being regarded by the older writers as nearly pathognomonic.

In order that the *arcus senilis* shall be significant of cardiac degeneration, the ring must be well defined, rather yellowish in color, the remainder of the cornea being cloudy and opaque, a tinge of jaundice coexisting.

Old people with fatty heart find their memory less retentive than in previous years, and those about them notice a change in their disposition; they are very apt to become morose, moody, irritable, and to have an habitual depression of spirits. Derangements of the digestive organs, anorexia, and sometimes diarrhœa and vomiting occur with senile fatty heart. The *pulse* is *feeble*, never strong, although it varies in force at different times: at one time regular, at another irregular in both rhythm and force. The irregularity is most apparent after sudden excitement or violent exertion. At times the pulse will beat very rapidly for a few moments, and then drop to thirty or forty per minute, becoming in such cases markedly irregular.

The rule is that, in old age, fatty heart is accompanied by a slow, weak, and irregular pulse.

A peculiar kind of dyspnœa, the "Cheyne-Stokes dyspnœa," or "the ascending and descending respiration," was formerly regarded as characteristic of fatty degeneration of the heart. This is not true, but it so frequently accompanies this disease that it is necessary to refer to it.

Cheyne, who first wrote about it nearly eighty years ago, thus describes it: "For several days the patient's breathing was irregular; it would entirely *cease* for a quarter of a minute, then it would become perceptible, though very slow. Then, by degrees, it became heaving and quick, and next would cease again. This revolution in the state of breathing occupied about a minute, during which there were about thirty acts of respiration."

Physical signs.—Inspection.—The apex-beat is seen faintly and indistinctly; in extreme cases it is invisible.

On *palpation* the hand will either detect no movement whatever over the precordial space, or the movements will be only occasionally felt, and then but very feebly. The impulse resembles that imparted to the hand by an aneurismal tumor.

When dilatation coexists, a tumbling, rolling motion will be noticed, but this is a sign of dilatation, *not* of fatty heart.

Percussion may elicit dulness over an abnormally large area, but this is from "obesity" or "fatty overgrowth" of the heart, or from pre-existing dilatation, which is so frequently associated with this form of cardiac degeneration.

Auscultation.—The first sound of the heart will be feeble or absent. When audible it is entirely valvular, and is followed by a comparatively long period of silence. It is a "toneless" sound, and is higher-pitched than is usual in old age. Indeed, the shortening of and rise in pitch in the first sound may cause it to closely resemble the second.

The second sound is feeble, and less distinct than the first. Rarely will you have murmurs in senile fatty heart, whatever may be the anatomical condition of the valves.

Temporary absence of the first sound may occur in *senile anæmia* and in some acute diseases of advanced life; but the sound will return when the anæmia disappears, or during convalescence from the disease. Thus, a continued absence of the muscular elements of the first sound becomes one of the most important signs of fatty heart.

Differential diagnosis.—Cardiac dilatation is most likely to be mistaken for fatty heart, and, indeed, the two often coexist.

In *dilatation*, percussion elicits a well-marked increase in the area of cardiac dulness; while in simple fatty degeneration the outline of the heart is shown to be normal. The first sound in dilatation may be feeble, but it is *never* absent, a condition frequently met with in senile fatty degeneration.

There are few, if any, of the cerebral symptoms in dilatation which so constantly attend a fatty heart. The Cheyne-Stokes respiration and the *arcus senilis* are prominent in fatty degeneration, and do not occur in dilatation of the heart.

Symptoms of pulmonic obstruction and congestion will rarely be absent in cases of dilatation which will be likely to be confounded with fatty heart, while such symptoms are exceedingly rare in cardiac degenerations.

Prognosis.—The prognosis in fatty heart is bad; it is incurable.

The fatal issue may be delayed when the cause can be removed, as in cases of chronic alcoholismus; some claim that a condition of fatty heart, accompanied by a like degeneration of the vessels, is not without its advantages, since a feeble heart is thus adapted to a feeble arterial system.

Fatty degeneration of the cardiac wall may exist for years, but life is always insecure; it is the fact that sudden death may occur at any moment which renders the disease so formidable.

These patients become more and more feeble until, finally, general dropsy sets in, and asthenia closes the scene; or more frequently syncope, coma, or rupture of the heart, unexpectedly cause the fatal issue. Rupture of the heart has no well-defined symptoms, and is immediately fatal.

Treatment.—Nothing can restore a heart in the advanced stage of fatty degeneration. The age of the patient, the feeble heart, the cachectic condition, all demand a supporting and a tonic plan of treatment.

Iron, strychnia, cod-liver oil, a generous but easily assimilated diet, a

moderate allowance of some nutritious wine, as Burgundy, are the only means which we have for increasing and improving the tissue-making power.

It is hardly necessary for me to say that when there is any palpable existing cause, such as chronic alcoholismus, it must at once be removed. No violent exercise, no prolonged moderate exercise, no excitement can be tolerated by these patients without absolute harm. The patient must live the life of an invalid, and all hygienic measures must be strictly regarded. The bowels must never become constipated, for straining at stool might cause rupture of the heart and death.

Digitalis is of little service; its administration, may, however, give temporary relief.

In simple *obesity* of the heart, alkalies are believed to have a very favorable influence, and here the food must contain a minimum of hydrocarbons, and if an excessive amount of food has been taken, the amount must be restricted. Exercise may be allowed to a moderate degree, but our main reliance is on the diet.

The attacks of syncope may be relieved by such stimulants as strong coffee and ether; the nitrite of amyl relieves both the fits of dyspnœa and the suffocative oppression accompanying anginal pain.

To relieve pain by narcotics is a dangerous procedure under these circumstances; hypodermics of morphia have been followed by instant death, as also have inhalations of chloroform; ether is perhaps the least dangerous.

The treatment is purely palliative, and the prolongation of life depends on the ability of the patient to so regulate his diet and surroundings, and to spend so much of his time in the open air as his particular case seems to require.

LECTURE XXVII.

CEREBRAL HEMORRHAGE.—APOPLEXY.

Summary.—Morbid Anatomy—Etiology—Symptoms—Differential Diagnosis—Prognosis—Treatment—Cerebral Softening—Ramollissement—Morbid Anatomy—Etiology.

GENTLEMEN:—By the term cerebral hemorrhage is understood an extravasation of blood within the cranial cavity. The term apoplexy, literally means the results produced by the extravasation of blood into brain-tissue; but it is often applied to those extravasations which are serous, as well as to those which are sanguineous. Hemorrhages, the result of traumatism, are not included under the head of apoplexy.

Morbid anatomy.—There are two varieties of cerebral hemorrhages: one consisting of minute blood-extravasations, or points; the other of clots of varying size.

Preceding either form, the vessel ruptured is the seat of miliary aneurism; these aneurismal dilatations upon the arteries are caused by a periarteritis, commencing in the lymph-sheaths of the vessels and advancing to the tunica adventitia and the muscular coat.

Minute blood-extravasations play an important part in the development of apoplexy. Little foci sometimes occur within the cortex of the brain as the result of venous thrombi, but these are probably soon absorbed. You may find them accompanying cerebral softening, and also in the neighborhood of large apoplectic spots. A congregation of these pin-head extravasations may constitute apoplectic foci.

At an autopsy we usually find irregularly spherical clots of blood, varying in size, but usually about three-fourths of an inch in diameter, imbedded in the cerebral substance.

In the aged, often a large portion of the brain is occupied by the extravasation.

The favorite localities for these hemorrhagic extravasations are the intraventricular nucleus of the corpus striatum, the extraventricular nucleus, the thalamus opticus, the cerebellum, and the pons. More frequently in the aged than in others will you find the corpus striatum pushed up and surrounded by the extravasation, instead of having an effusion into it; this is best seen in the ventricles, which may also be the seat of hemorrhage, sometimes so extensive that their septa are torn and blood escapes upon the surface of the brain.

In the aged you will quite often notice apoplectic foci between the membranes in the subarachnoidean space, or even superficially. The locality of the extravasation is determined by the arterial distribution, as the vessels to those parts already named are the most direct continuation of the carotids.

The cerebral convolutions are flattened and the sulci deepened when

the extravasation is extensive; the dura mater is stretched, and sometimes there is a visible bulging, for, in advanced life, large hemorrhages are rather the rule. The clot, or "focus," is a soft, grumous mass, at whose centre is the opening into the ruptured vessel. Its wall consists of shreddy, cerebral matter, mingled with fibrinous fibrillæ, the result of the hemorrhage.

When the apoplectic stroke, as it is called, is not immediately fatal, the clot may undergo three changes: 1st, its fluid may be absorbed; 2d, it may undergo fatty degeneration and be absorbed; 3d, it may excite inflammation in the surrounding brain-substance, and lead to red softening. In all cases the brain-substance in the immediate vicinity of the clot is somewhat œdematous, and slightly yellowed.

The secondary changes consist in the transformations of the clot into a *cyst*, whose contents vary in color from a pale yellow to a dark brown, according to the number of blood-corpuscles that undergo pigment degeneration. The cyst-wall is firm, smooth, connective tissue. A cyst rarely becomes an abscess of the brain in the aged.

Cicatricial tissue may form from the cyst, although it is, at present, a mooted question whether a cyst must necessarily pre-exist in order that such a cicatrix shall form. It takes about two years, in old people, for such a cicatrix to form and the cyst to be absorbed. There may be a number of these cysts or cicatrices in the same brain, corresponding to the number of apoplexies. Cruveilhier states that he found fifteen in one brain. They cause "puckering" of the brain-substance.

The nerves connected with the parts involved may undergo degeneration, and general atrophy of the brain may ensue.

Etiology.—The most powerful predisposing cause to cerebral hemorrhage is *age*. It rarely occurs before forty, the liability to it increasing with advanced years. It is generally stated that the tendency ceases at about seventy; but this is not so, for the comparatively small number of people living over seventy has not entered into the statistical estimate.

The causes which *predispose* to apoplexy are fatty, atheromatous, or fibroid degeneration of the walls of the vessels; hence, the importance of gout, rheumatism, syphilis, alcoholismus, and chronic Bright's disease as predisposing causes.

Cardiac hypertrophy and dilatation predispose to apoplexy; aortic insufficiency and pulmonary emphysema are also important etiological factors.

Softening of the brain, which, I think, follows as often as it precedes apoplexy, also predisposes to it.

Men are far more liable to cerebral hemorrhage than women, on account of their active mode of life and their greater liability to excitement. Apoplexies also occur much more frequently in cold than in warm weather.

The *exciting* causes may be summed up in the phrase, *increase of blood-pressure;* although a cerebral hemorrhage may occur without any such increase, at least so far as we can discover. Coughing, running, a fall, violent emotion, the venereal act (the latter especially) are frequently exciting causes of apoplexy in the aged. A cold bath, by constringing the cutaneous vessels, may be an exciting cause.

The so-called "plethoric habit," which causes in some so much anxiety, has no significance, for the emaciated valetudinarian is just as liable to apoplexy as he of the opposite condition.

There is in certain persons an hereditary tendency to arterial degeneration which predisposes to cerebral hemorrhage.

Symptoms.—There may or may not be prodromata of a cerebral apoplexy. When the latter are present they may appear either in the form of

vertigo, muscæ volitantes, double vision, temporary blindness, tinnitus aurium, complete deafness, an abnormally keen sense of smell, or a total loss of it. These are, however, unimportant, compared to loss of memory, difficulty of speech, lethargy, or stupor, and a feeling of weight, numbness, or formication that very commonly presages a fit of apoplexy, and which should always excite alarm when present during the senile period of life. Repeated epistaxis is another important prodromal symptom.

These premonitory signs may be absent, and the attack be instantaneous, the patient suddenly falling into a condition of coma, with loss of sense, sensation, and voluntary motion; or, the coma comes on gradually, being preceded by pains in the head and a feeling of faintness; or, finally, aphasia and hemiplegia are the first and the only symptoms.

The attack may be so light as to seem nothing more than a momentary state of insensibility; and the patient is thought to have had "a fainting-spell" or "a fit of indigestion," since it frequently comes on after an aged person has been over-indulgent at table.

Usually, however, the individual is struck down suddenly, and there is complete loss of consciousness for a period varying in length from a few minutes to two or three days.

During the state of coma the respirations are deep, slow, and often stertorous; the face is either pale, or red and turgid; the veins are engorged, and as the coma deepens the face assumes a dark, livid hue. A marked pallor may sometimes continue through the whole attack. If the coma last three days, the temperature on the second day is lowered (96° F.) frequently to rise the next day to 107° F. The pulse in old people at the onset of the "fit" is commonly rapid, feeble and intermittent; later it becomes slow, full, and regular. As a rule, the pupils are dilated, though at times, while one is of normal size, the other is dilated or contracted.

In its worst form—"*apoplexie foudroyante*"—the breathing is exceedingly slow, stertorous, often superficial, interrupted, and irregular. Each expiration is accompanied by a loud "puffing" noise from the lips, and the ejection of a frothy mucus which accumulates about the mouth. Deglutition is impossible; the features are distorted, the pupils contracted, the skin is cold and clammy, and the fæces and urine are passed involuntarily. Death usually occurs, in such cases, in two or three hours. Reflex movements may be excited, except in very rare instances. The paralyzed side is usually convulsed from the beginning, and tetanic spasm of a muscle, or set of muscles, occasionally occurs.

There is a form of apoplexy which is sometimes observed in the aged, seemingly associated with *ventricular effusion*—a general convulsive attack, epileptic in character, where the tongue is often bitten and frothing at the mouth occurs, lasting from fifteen to thirty minutes, which is followed apparently by no bad results other than the gradual supervention of extreme debility. But death almost always ensues after a longer or shorter period varying with the age and constitution of the patient.

Apoplexy in the aged, is synonymous with hemiplegia, and its evidences are too well-marked to need any lengthy description. The "conjugate deviation" of the eyes, the protrusion of the tongue toward the paralyzed side, the drawn mouth, etc., these are the common symptoms. Anæsthesia on the paralyzed side commonly occurs, but passes off gradually in most cases.

The hemiplegia itself is permanent or temporary, depending on complete destruction or only partial implication of the corpus striatum or thalamus opticus.

I shall not attempt to name all the variations due to hemorrhages into the pons, cerebellum, etc.

Differential diagnosis.—Cerebral hemorrhage in the aged may be mistaken for cerebral congestion, uræmia, alcoholic intoxication, and embolism.

In *congestion* there is absence of stertorous breathing, which is always present in apoplexy.

The pupils are contracted in congestion, and dilated in apoplexy.

The coma is of very short duration in congestion, whereas it persists for some time in apoplexy.

Congestion has a long prodromal period; while it is short, and often absent, in cases of apoplexy.

In congestion the paralysis is usually bilateral, while in apoplexy it is unilateral.

Hemiplegia is rarely present in uræmia, while it is rarely absent in apoplexy.

Uræmia comes on gradually, and is usually preceded by convulsions; while the coma of apoplexy is sudden in its advent, and convulsions never precede it.

The presence of casts and albumen in the urine establishes the diagnosis between uræmia and apoplexy.

Alcoholic intoxication is often mistaken for apoplexy. The patient can easily be roused from alcoholic coma, while this is not the case with cerebral hemorrhage.

There is no stertor in alcoholic coma, whereas it is usually present in uræmia.

The pulse is feeble and frequent in alcoholic coma, while it is full, strong, and slow in apoplexy.

There is no hemiplegia in alcoholic coma, while it is an exceedingly common condition in apoplexy.

Lastly, the urine may be tested for alcohol as follows: to fifteen minims of a solution consisting of three hundred parts of strong sulphuric acid, and one of bichromate of potassa, add a few drops of the suspected urine; in case of alcoholism, the mixture turns an emerald green.

Cerebral embolism rarely occurs in the aged, and is accompanied by a history of rheumatism or rheumatic endocarditis and valvular disease of the heart. In embolism, the patient does not lose consciousness; in apoplexy there is loss of consciousness. The pulse is rapid and feeble in embolism, and the face is pallid; in apoplexy the pulse is slow and full, the face is red and turgid. Aphasia is rare in apoplexy, while almost pathognomonic of embolism.

The pupils are unaltered in embolism, while in apoplexy they are usually deviated from the normal.

There is stertor in apoplexy, whereas in embolism the breathing is normal. Paralysis is usually on the right side in embolism, while it is on either side in cerebral hemorrhage. Evidence of arterial atheroma is usually present in apoplexy, it may be absent in cases of embolism. The paralysis improves in forty-eight hours, in embolism, while in apoplexy it is of much longer duration, and is rarely entirely recovered from.

Prognosis.—The prognosis in the apoplexy of old age is always grave. If the first attack is recovered from, it is almost always followed by a second within a year; a third attack is almost always fatal.

The greater the age, the greater the danger.

Persistent coma, loss of control over the sphincters, dysphagia, "pinhead" pupils, a temperature over 100° Fahr., and a "puffy" expiration, are

all unfavorable symptoms ; and convulsions, indicating that the hemorrhage has involved the meninges, are generally soon followed by death.

The general condition of the patient, and his freedom from disease, will likewise influence the prognosis. Never give a positive prognosis until at least two weeks have elapsed after the attack.

Even after so-called recovery has taken place in the aged, there is more or less loss of mental power, so that the patient is incapable of transacting business accurately, and even the power of reading, or the memory of certain words, is wholly lost. The more complete the paralysis, the less the chance of recovery.

Treatment.—The *prophylactic* treatment resolves itself into avoidance of sudden violent physical exertion, or of strong emotion. The diet should be simple and non-stimulating. The clothing should be worn loosely, and there should be free ventilation in the living and sleeping apartments.

Sudden extremes of temperature should be avoided ; hence, hot and cold baths are contraindicated. Attention must be paid to the bowels, and, in the advent of premonitory symptoms, active purgation and blisters to the neck will be found of service.

Alcoholic beverages in *excess* are most harmful, but light wines, in moderation, do no injury.

The bromides of lithium and zinc oxide are often administered with advantage when cerebral hemorrhage threatens. Finally, remember that you are dealing with old age, and that the patient must not be debilitated.

When an attack or "stroke" *has occurred*, the first things to be done are to elevate the patient's head, loosen the clothing about the neck, and have the apartment kept dark and absolutely quiet. The air about the head may be kept cool by ice-bags, and the feet are to be placed in a hot mustard-bath.

Under no circumstances should bloodletting be practised in the treatment of apoplexy of old age.

The bowels should be moved by drastic cathartics, sufficiently to induce the so-called "revulsive" effects.

Much of the venous turgescence of countenance may be due to stertor from falling back of the tongue ; hence, place the patient on his side, and it will frequently disappear. Emetics are never to be administered. Sinapisms may be applied to the nape of the neck, calves of the legs, and over the stomach, their size and number being determined by the condition and age of the patient.

When the vital powers are greatly depressed, when there is extreme feebleness and pallor, internal and external stimulation must be resorted to ; milk, beef-tea, and brandy are to be given freely. Iron can be administered to the aged in nearly all cases of cerebral hemorrhage. The "hydropathic" treatment is now seldom resorted to for the hemiplegia which follows apoplexy in the aged. Mild narcotics are to be given in case there is much sleeplessness and irritability, but only after a lapse of two or three weeks.

When all symptoms of cerebral irritation have passed, faradization may be employed ; and phosphorus and strychnia (the latter hypodermically) may be given with advantage.

I now invite your attention to the study of senile softening of the brain.

Cerebral Softening.

Cerebral ramollissement, or encephalomalachia, is a disease peculiar to old age. It is one of the most frequent cerebral diseases of advanced life.

Morbid anatomy.—This condition has, in reality, been studied only within the present century; and many theories have been advanced as to its nature and pathogenesis. Some claim that it is always the result of inflammation; others say it is a variety of gangrene; and still others, that it is the result of a chemico-pathological process.

Three forms are discoverable at the autopsy: the red, the yellow, and the white softening. The red has been called acute or inflammatory; and the white, chronic or non-inflammatory softening, though this distinction cannot always be demonstrated.

I.—*Red softening* is first marked by stasis, rapidly followed by fatty degeneration of the cells and nerve-fibres. The pultaceous tissue is of a deep red color, or the discoloration may occur in spots as numerous foci in different stages of softening, the processes in either case being best marked at the centre of the softened mass or masses. The vessels are filled with coagulated blood, their contents undergoing a retrograde metamorphosis, and the fibrin becoming granular, the infarction becomes dry and shrunken, cicatrization may occur, or there may be liquefaction of the contents, and the formation of cysts. Increase in specific gravity in red softening occurs in a few instances, although decrease is the rule.

The microscope reveals the presence of fat-granules, altered blood-corpuscles, and large granular corpuscles (*Gluge's corpuscles*) from the cells of the neuroglia. The capillaries are dilated and filled with coagula, and the white substance of the fibres is coagulated or broken up into large masses of myeline.

This variety of cerebral softening is properly called a *necrobiosis;* its color is due to its sudden occurrence, the subsequent pathological changes *being common to all three forms.*

II.—The softening from anæmia, *i.e.*, from obstruction in the vessels, is called *yellow softening.* It may occur at any point in the brain, but its most frequent seat is in the middle or posterior lobes, and in the convolutions or corpus striatum. It is often the color of sulphur, varying in size from that of a hazel-nut to that of the fist; it may involve a whole hemisphere. Its consistence varies, but in typical cases it is a gelatinous, moist, and tremulous pulp. Later on the so-called "cellular infiltration" of the French writers occurs: the implicated spot is metamorphosed into a mass of reticulated fibres, in whose meshes is a milky, chalky fluid. This is a sort of reparative process, having its analogue on the surface of the brain in the yellow plaques made up of tough, pliable, ochre-colored connective tissue containing nuclei, crystals of hæmatin and hæmatoidin, and a few fat-corpuscles. When cut across, one of these spots rises above the level of the section; a gentle stream of water will wash away the softened tissue from the surrounding cerebral substance. In all varieties of softening there is usually no well-marked line of demarcation; the healthy and diseased parts insensibly run into each other. In the yellow variety we can occasionally find traces of demarcation.

The color of yellow softening depends on a closer congregation and a finer subdivision of the fat-globules than occurs in white softening, though changed blood-pigment sometimes gives it its yellow color.

III.—*White*, atrophic, or chronic *softening* is the variety most frequently met with in the aged.

It is white, or resembles healthy brain-tissue, for the reason that the process takes place slowly, and hence hyperæmia or hemorrhage is very slight or wholly absent; often the implicated spot is as diffluent as cream. The other pathological appearances are the same as in yellow softening, with which it is identical, except in color.

There is a decrease in the specific gravity of brain-tissue that has undergone white ramollissement.

Etiology.—I have said that the difference between red and white or yellow softening, as marked by the terms acute and chronic inflammatory and non-inflammatory, is not exact. Its etiology will explain this.

Encephalomalachia is frequently the result of embolism, thrombosis, or hemorrhage. And since almost all the predisposing causes of thrombosis are met with in advanced life, thrombosis is by far the most frequent cause. It sometimes results from syphilitic disease of the arteries.

It is quite well established that in advanced life severe and prolonged intellectual efforts or exercise of the emotions will cause cerebral softening; blows, or the action of intense cold on the head, and alcoholismus act also as causes.

Atheroma in predisposing to thrombosis is a powerful factor, and the pressure of intracranial tumors has been known to produce ramollissement. Cardiac valvular diseases exert a most powerful influence in the development of cerebral softening.

The liability to cerebral softening steadily increases with advancing age.

Red softening may be caused by embolism or thrombosis, and is then sudden; or the vessels in chronic white softening may rupture, and the blood extravasated give a red color to the softened mass.

Yellow softening may be, and commonly is, as I have said, a variety of white; in some instances it primarily results from embolism or thrombosis; a gelatinous œdema about cerebral neoplasia has been denominated a yellow softening.

White softening may in rare instances be acute, from a sudden obstruction by an embolus in one of the larger arteries; it may be due to the senile change in the vessels, and it may result from feeble heart-power. In some instances there is no discoverable cause.

At my next lecture I shall speak of the symptoms and diagnosis of cerebral softening.

LECTURE XXVIII.

CEREBRAL SOFTENING.

Summary.—Symptoms—Differential Diagnosis—Prognosis—Treatment—Senile Cerebral Atrophy—Morbid Anatomy—Etiology—Symptoms—Differential Diagnosis—Prognosis—Treatment.

Anatomical and Physiological Changes in the Alimentary Canal in Old Age.

Chronic Senile Gastric Catarrh (Dyspepsia)—Morbid Anatomy—Etiology—Symptoms—Differential Diagnosis.

GENTLEMEN:—In *acute cerebral softening* prodromal symptoms frequently exist. These premonitory signs are similar to those of apoplexy; although formication, aching, and cramps of the limbs, and anomalies in the sense of touch, are much more prominent in commencing ramollissement. Local inequalities of temperature are occasionally among the prodromata. There is also a diminution in the motor power; when this is present, it is an important sign. When prodromata occur, the symptoms either gradually increase, or else advance by sudden exacerbations with intervals of apparent improvement—the affected side grows weaker and weaker, the fingers, hands, and feet are managed clumsily, the leg trembles under the body, there is a tottering gait, and, finally, decided paralysis.

It now either assumes the "chronic" form, or typhoid symptoms come on, and death speedily occurs, often from complications in the respiratory organs; the older the patient, the less likely are we to have premonitory signs. Often the attack simulates cerebral hemorrhage so closely that a differential diagnosis is impossible.

In acute cases, *without* prodromata, the patient becomes suddenly paralyzed without loss of consciousness, or is paralyzed and comatose; but he soon comes out of the coma with *impaired or lost* speech. Vertigo is a very constant symptom, and when headache is present on the side opposite the paralyzed one, it is almost diagnostic.

In some cases a mild delirium, a "wandering" as it is called, of short duration may mark the onset; this occurs most frequently in very old persons. The eyeballs are directed to one side, although the features are symmetrical until active movements are made.

Muscular rigidity, spasmodic twitching, difficult deglutition, suffusion of the eye, are usually present; the urine and fæces are passed involuntarily, bedsores form, the pulse grows more rapid and feeble, and the patient finally dies from exhaustion.

Chronic softening is for weeks or months preceded by gradually increasing feebleness, loss of memory, fretfulness, and fits of uncontrollable weeping—rarely laughing. There are dull pains, or "a sense of confusion," in the head; there is general loss of muscular power, with difficulty or "*thickness*" *of speech*. The patient becomes perfectly listless and there is a marked change in disposition. Then comes partial paralysis of one side, and finally the patient becomes perfectly helpless and childish, and

he is incapable of retaining the contents of his bladder and rectum. Aphasia, from plugging of the left middle cerebral artery, is a symptom which frequently accompanies chronic softening. When paralysis begins at the distal end of a limb and approaches by degrees the trunk, it is called "*creeping palsy.*"

The intellect is much oftener affected in chronic than in acute ramollissement, and febrile symptoms are exceptional occurrences.

The bowels are commonly constipated, the appetite and the body weight usually remain at the normal standard, and the patient is disposed to sleep the greater part of his time. In some cases there are no symptoms to mark its presence.

Differential diagnosis.—The differential diagnosis between cerebral softening, apoplexy and embolism we have already considered.

In some instances the symptoms which attend the development of tumors of the brain may be mistaken for those of cerebral softening.

The history, in cerebral softening, usually elicits cardiac disease or atheromatous arteries; while carcinoma and gummata, the most frequent senile cerebral neoplasia, will probably have a history of syphilis or cancer.

Localized pain in the head is a prominent and almost constant symptom in cerebral tumor, while the headache is dull and diffused, often absent, in softening—pain is not a necessary symptom of softening; this is one of the chief points of its diagnosis.

The speech and intellect are unaffected in tumors; whereas both are *early* implicated in cases of softening.

Symptoms referable to some special cranial nerve or set of nerves are present in cases of cerebral tumors, and absent, as a rule when softening exists. The *face* is prominently implicated in the paralysis from tumors, while hemiplegia is the rule with softening.

Epileptic convulsions, independent of paralysis, occur frequently with tumors, but they do not exist in cases of cerebral softening.

If tumors are syphilitic, the symptoms which they give rise to improve under large doses of iodide of potassium, while this drug has no effect in cerebral softening.

Prognosis.—Senile cerebral softening is regarded as inevitably fatal. In acute softening death has often occurred on the first day, and life is not prolonged over ten days.

Chronic cerebral softening is very varying in its duration; cases have been reported where the symptoms were well marked and extended over a period of four years.

Death may directly occur from the softening, either from interference with respiratory apparatus, or from the complications, such as bronchitis, pneumonia, meningitis, hypostatic congestion of the lungs, diarrhœa, extensive sloughs on the sacrum and hips, hemorrhage into the softened mass, or from exhaustion.

Treatment.—When the premonitory symptoms of cerebral softening occur the strictest attention must be paid to the diet, which must be simple, nutritious, and easy of digestion. The surroundings of the patient should also be regarded, and no excitement or over-exercise allowed. These patients should never be allowed to become constipated. Milk is the best article of diet in these cases.

When there is excitement followed by sleeplessness or headache, the bromides may be administered; and Indian hemp is regarded by some as a most useful drug in this condition.

When softening is established, benefit is often obtained from zinc, phos-

phorus and strychnia, and the tonics iron and quinine are beneficial in nearly every case. The *constant* current, alternating with faradization, is to be given with great care.

When cerebral softening simulates apoplectic seizures, the same recumbent posture, cool surroundings, quiet, etc., must be preserved as were advised in cerebral hemorrhage, and the bowels must never be allowed to become constipated, you will find this to be the tendency in the vast majority of cases. When bedsores and sloughing threaten, they must be promptly combated, for the strength of the patient is greatly diminished by them, and in the aged they are very obstinate and often cause death.

Blistering, counter-irritation and bloodletting are contraindicated in all cases.

The last senile cerebral disease which I shall consider is

Senile Cerebral Atrophy.

Morbid anatomy.—In senile atrophy there is actual diminution in the cellular elements of the brain, and a loss in its interstitial connective tissue. The cells of the cortex are swollen and pigmented, and pigmentation also occurs in the walls of the vessels which are commonly the seat of fatty degeneration. Corpora amylacea are present in the vast majority of cases, especially in the thinned cortex. In general it may be said there is decrease in the fat, and increase in the water of the brain-substance.

Senile atrophy is usually complete; when partial it affects the left hemisphere. There is unequal thinning of the convolutions, and the sulci are larger and deeper. There is increased consistence, often toughness, of the brain, the meninges are clouded, and the ventricles are dilated with fluid, varying in amount from two to twelve drachms; this must be regarded as a purely *conservative* process. Serum likewise fills the subarachnoidean space.

An attempt to cut the brain shows it to have a leathery toughness, and the section is frequently corrugated: the medullary portion will have a dull white or drab color, and the cortical substance will be darker than normal.

Cases occasionally are found where the arachnoid covering the hemispheres is in juxtaposition with that of the ventricles, *there being no nerve-substance between;* again, only a thin layer of areolar tissue may intervene between these two membranes.

Etiology.—Atrophy may follow a hemorrhage or softening, and is occasionally caused by tumors and inflammation of the coverings of the brain. Injury or destruction of the peripheral nerves may induce secondary cerebral atrophy. But the great cause is senile marasmus.

Symptoms.—The mental faculties in senile cerebral atrophy gradually become weaker and weaker; there are loss of memory and blunting of the special senses; and the movements, from being unsteady, are soon accompanied by tremor.

The patient becomes childish, apathetic, and is constantly sleepy or asleep, and nearly all control over the sphincters is lost.

The disease finally ends in invasion of the medulla, implication of the centres situate therein, and deglutition or respiration, or both, are so much interferred with, if not annulled, as to cause death.

It may be that general paralysis of both sides of the body occurs, and then complete imbecility usually supervenes.

Differential diagnosis.—This is made from softening and hemorrhage only, by exclusion and a careful consideration of the history.

Prognosis.—This is always unfavorable; no estimate can be made as to the length of life, and the intellect and forces cannot be rallied by any known means. Its duration is uncertain.

Treatment.—Treatment is out of the question; we can only build up the patient by means of a good diet and tonics.

I shall now pass to the consideration of senile diseases of the *digestive apparatus*. The most common and important of this class is

Chronic Senile Gastric Catarrh.

The digestive apparatus participates in the general atrophy which occurs in all the tissues in old age. The stomach and intestines lose bulk, and their mucous membrane is markedly thinned.

The glandular system is more or less atrophied, and many of the glands seem to have entirely disappeared. Not only is the gastric mucous membrane thinned, but it is also paler than in adult life, usually acquiring an ashy gray color, which is more pronounced the older the individual.

The vessels of the mucous membrane, the veins especially, are dilated and varicose. The glands in the stomach are occasionally so impregnated with melanin that they have a punctated look, resembling the cutaneous surface when filled with specks of gunpowder. Numerous small yellow spots (groups of glands filled with fatty matter), occur in the gastric mucous membrane in that condition known as senile marasmus. The duodenum, jejunum and ilium undergo more extensive wasting of their coats than occurs in the stomach; indeed, these structures are sometimes so attenuated that at the autopsy their contents are distinctly visible through their walls.

It may be mentioned that the large intestine does not exhibit so much wasting, since there is more or less compensatory hypertrophy of its muscular coat.

Atrophy of the jaws, the lower especially, results from loss of the teeth, a common condition in old age; and even on the surface of the tongue we find that thinning of the digestive mucous membrane already referred to.

The *physiological* results of these conditions are a blunting of the sense of taste, more or less dysphagia, and difficult mastication; a slow and imperfect stomach and intestinal digestion.

As old people lose their teeth and their power of mastication, they consume less bread and meat, as well as other solid food; hence, chronic anæmia becomes a physiological condition in many old people. The result of all these changes is a diminution in the absorption of nutritious elements.

In this connection, it may be stated that in old age the bile is much richer in cholesterin than in adult life. The salivary glands, the pancreas, and the mesenteric glands share in the general glandular atrophy.

Senile gastric catarrh, known also as chronic gastric catarrh of the stomach, catarrhal gastritis, and follicular dyspepsia, is very frequently met with in advanced life.

Morbid anatomy.—You find, on examination of a stomach which is the seat of these changes, no such thick layer of mucus as is present in the gastric catarrh of adult life; it must be remembered that the secretion of mucus is very much diminished in old age, both as a result of the senile

state, and also as a result of diseased process. Some mucus, however, is found in every case. There are sometimes ecchymotic spots on the membrane, the result of hemorrhages from the varicose vessels.

The color of the mucous membrane varies; sometimes it retains its natural color, but oftener it assumes a reddish brown, slaty gray, or dirty blue appearance.

The membrane is unevenly thickened, tougher than normal, and is very loosely connected with the cellular tissue beneath. In old age it is often corrugated, or thrown into furrows; and villous projections one-fiftieth of an inch in height, filled with granular corpuscles, are found near the pylorus.

Mucous polypi are almost exclusively limited to the stomachs of *old* people with chronic gastritis; they are either sessile or pedunculated, and their nature and consistence vary according as they are composed of fibrous-tissue, papillæ cysts of the obstructed glands, or of all these united.

Ulceration, thickening, and induration of the diseased membrane is of frequent occurrence, the ulcers being superficial, limited to the mucous coat, and, by their aggregation, a large extent of the membrane may suffer abrasion.

When the disease has existed for a long time, the mucous membrane has a granular or mammillary appearance which results either from obstruction to the exit of the secretion from the tubules (thus causing them to stand out on an atrophied tissue), or mammillation may result from actual *hypertrophy* of the glandular layer. The microscope may show the tubules to be completely filled with fat-globules, or the epithelium to have undergone fatty degeneration.

In other cases the tubules will be found filled with a granular detritus, and their epithelium will have undergone granular degeneration, which at the base is sometimes darkened by blood-stains.

In whatever condition their contents may be, the *mucous follicles in senile chronic catarrh are always enlarged*, sometimes visibly so. By many these are regarded *as the essential seat of the disease*. In long-continued gastritis the submucous coat becomes involved, and more or less thickening and congestion occur in it. If there is an infiltration into this layer, its organization and subsequent contraction may hinder still more the peristaltic motion already impeded to a greater or lesser extent by the senile intestinal changes already referred to.

In many old people who have for years suffered from this disease, you may even find the muscular coat involved in the process of thickening, and this additional pathological lesion will reduce peristalsis to a minimum.

Etiology.—Undoubted examples of primary acute or subacute gastritis are seen in old age. These are *very* rare, however, at this period of life, and may be thrown out as etiological factors, senile gastric catarrh being *chronic* from its commencement.

Catarrhal dyspepsia is in old age very frequently associated with, and appears to be secondary to chronic bronchitis, bronchorrhœa, and chronic disease of the heart. One very important cause is anæmia; the senile anæmic condition which I have already referred to.

Long-continued, passive hyperæmia, the result of obstruction to the portal circulation and pressure from tumors, acts as a producing cause, though not as frequently as chronic bronchitis and organic disease of the heart. Many old people will give such a clear history of ancestral dyspepsias that there is no doubt but that the disease is sometimes hereditary.

Old people who habitually eat too rapidly or too much, or who eat large quantities of food which in youth their stomach could manage, but

which, at this period of life, is utterly indigestible, always have embarrassed digestion, or, as they prefer to call it "a *morbidly sensitive* stomach." Again, unless the greatest care be taken, even slow and moderate eaters will, when their teeth drop out or decay, suffer from this painful condition on account of insufficient mastication. Immoderate smoking or snuff-taking may, in advanced life, induce catarrhal dyspepsia.

Finally, alcoholic beverages, too little exercise, mental strain or physical labor immediately after taking food, and irregularity in the time of eating, are very often followed by "dyspeptic" symptoms in the senile period of life. Scrofula, syphilis, and gout, undoubtedly predispose to, and the latter actually excites senile chronic gastritis.

Symptoms.—In old age, you will find that chronic catarrh, or, as it is usually called, "dyspepsia," appears in a variety of ways. Some old people say their stomach was always weak, or that certain articles, mentioning those which they eat most of, always disagreed with them. Others, again, are truly great sufferers, and in them the condition amounts to more than an infirmity of advanced life. There is generally a sense of weight and fulness in the region of the stomach, either constant, or coming on in from one-half to an hour after the ingestion of food.

Anorexia is present in nearly all cases of well-developed senile gastric catarrh, and the only food that is relished is that of a piquant and highly seasoned kind; they cannot bear oily food. This anorexia soon leads to a well-marked anæmic condition, and to an exhaustion which is far more dangerous than the emaciation accompanying it; thus, anorexia must be looked upon as a serious symptom, and one to be promptly combated. Accompanying the anorexia there is commonly great thirst, especially for acid drinks.

Old people with catarrhal dyspepsia vomit, or rather regurgitate, every morning, or at some other period of the day, more or less glairy mucus, just as bronchitic individuals "raise" phlegm of a morning on rising. These old people can, or do, regurgitate this mucus, and yet retain the contents of the stomach perfectly well. Nausea is far from infrequent in these cases, but very rarely is accompanied by vomiting.

Flatulence and heartburn are so common in many old people as to be regarded as the natural attendants of old age. *Heartburn* or cardialgia arises from the acid mucus which is belched up into the œsophagus.

Flatulence is sometimes the chief sign of this condition and is always very distressing; it is most annoying during or a little after the period of digestion, though you will find some old people complaining of it when the stomach is empty. Old gourmands form the greater part of the former, and chronic tipplers of the latter class, for self-evident reasons. Flatulence is sometimes so great that the distended stomach and intestines feel "sore," and are painful to the touch.

Pyrosis, "water-brash," or *black water*, as it is variously styled, is often present in old people, in women especially, as a chronic and obstinate accompaniment of catarrhal dyspepsia. The fluid is thin, watery, colorless, and either insipid to the taste or so acrid and sour that it sets the teeth on edge. More or less pain precedes the gush of liquid into the mouth, which is followed by almost immediate relief. You will find pyrosis commonest in old people who are ill-fed, poorly clothed, and who live principally upon vegetable food, or are habitual spirit-drinkers. Pyrosis may occasionally be so great as to amount to *gastrorrhœa*, a condition analogous to the blennorrhœa of other parts.

Gastrodynia, more or less severe pain in the stomach, is by no means an

infrequent symptom of senile gastric catarrh, and it is met with as well when the stomach is empty as during the various periods of digestion.

Sometimes the pain is diffused, and again it is confined to a spot no larger than a silver dollar; it is relieved by pressure.[1]

The *tongue* is usually pale and flabby in old subjects who have long suffered with gastric catarrh; often it is indented with the marks of the teeth. It may be studded with minute aphthous ulcers. The tongue in other cases has a peculiar "sodden" look, or may be perfectly normal even in very obstinate and prolonged cases. The breath is always offensive in these old patients. The texture and color of the skin, hair and nails are altered from the normal, and present a shrivelled appearance.

Old people who have long suffered from catarrhal dyspepsia are rarely free from hemorrhoids; they are also habitually constipated, intercurrent attacks of diarrhœa being, however, far from infrequent, and you may find large quantities of mucus mixed with scanty alvine dejections.

The *urine* is scanty, dark-colored, sometimes depositing urates, phosphates and oxalates, although it is far from being as sedimentary as in adult life. It is often clouded, even at the time of emission. The specific gravity varies with the diet and the time when voided, as for instance, whether morning or evening.

Finally there is a train of symptoms called "sympathetic," which occur late in the disease, and undoubtedly arise from the gastric disturbance.

Hypochondriasis, despondency, mistrust of old friends, and irritability of temper are common in these cases; there is either sleeplessness, or the sleep is disturbed. Dyspnœa, sighing, chilly sensations about the extremities, slight febrile reactions, preceded by rigors, slight night-sweats and an icteroid hue of the conjunctivæ, these are rarely absent in cases of senile gastric catarrh.

Differential diagnosis.—Chronic catarrh of the stomach, or catarrhal dyspepsia, may be mistaken for *ammonœmia* and *atonic dyspepsia.*

The points in the differential diagnosis between senile gastric catarrh and ammonæmia will be considered under the head of Ammonæmia.

Atonic dyspepsia occurs in those whose pursuit or mental condition is accompanied by great depression, whereas catarrhal dyspepsia is associated with chronic cardiac, pulmonary or hepatic disease, or is induced by "tippling." There is no pain or tenderness over the epigastric region in atonic dyspepsia, while these are never absent in senile chronic gastritis. The tongue is pale, broad, and flabby in atonic dyspepsia, and often covered with a thick coating in chronic gastric catarrh.

Anorexia and thirst are marked symptoms in senile chronic gastritis, while thirst is not a prominent symptom of atonic dyspepsia, and the appetite is slightly, if at all altered.

In atonic dyspepsia there are no constitutional or sympathetic symptoms, while progressive emaciation, weakness, a jaundiced, sallow, or earthy color to the skin, a well-marked cachectic state, and disordered mental condition, are prominent in chronic gastric catarrh.

The alterations in the urine of chronic gastritis, which I have just given, are not found in atonic dyspepsia, the secretion being normal.

Finally, the bowels are regular in atonic dyspepsia, while in senile gastric catarrh they are constipated, slight attacks of diarrhœa occurring at varying periods in the course of the disease.

I shall continue this subject at my next lecture.

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[1] The subject of *gouty gastritis* has been referred to by Professor Charcot in previous pages of this book.

LECTURE XXIX.

CHRONIC GASTRIC CATARRH.

Summary.—Prognosis—Treatment.
Diarrhœa in Old Age—Etiology—Symptoms—Varieties—Differential Diagnosis—Prognosis—Treatment.
Constipation in Old Age—Etiology—Symptoms.

GENTLEMEN:—The *prognosis* in the catarrhal dyspepsia of old age is always good; it is rarely a direct cause of death.

The prognosis for recovery depends on the age; when over seventy, if it has existed a considerable time, it is incurable. It also depends on the power or will of the patient to conform to the diet and regimen prescribed, and finally on the causative or complicating diseases. You will, therefore, carefully study the thoracic and hepatic conditions of an old person with chronic gastric catarrh, before giving what should always be a guarded prognosis.

Treatment.—All medical treatment is secondary to diet and regimen.

Abernethy said that a man could not be induced to attend to his digestive organs till death, or the fear of death, stared him in the face.

Indeed, in many instances a cure can be brought about by regulation of the diet alone, when there is no organic disease complicating the catarrh. When complications exist, the treatment resolves itself into the removal of the cause. This effected, the disease becomes readily amenable to treatment.

A diet for an old person with chronic catarrhal dyspepsia—subject of course to such variations as shall be indicated by the patient's idiosyncrasy—is the following:

First, it must be remembered that the greatest regularity as to the time of meals must be preserved; the number of the meals should be four or five, rather than three. Five or six hours must intervene between the times of taking food, to give the stomach its needed repose, and the popular error of thinking that, as age advances, the *quantity* of food must be increased, should always be avoided, for excess in old age means disease.

On rising, the patient should always take a glass of fresh milk, containing either soda- or lime-water; and when the strength and age do not contraindicate, a "rub-down" with a coarse towel will be found to have a wonderfully refreshing effect, and the relation between a brisk feeling of well-being and the appetite is too well known to need comment.

At breakfast the aged patient may have weak tea or milk, fresh eggs lightly cooked, a chop not too well done, stale bread with but little butter.

Luncheon may consist of oysters, or, when no meat has been taken at breakfast, of a chop or piece of mutton, a glass of old sherry, with perhaps an egg in it, and yesterday's bread.

Or the luncheon may be some fresh broth, a sandwich of grated fowl, a glass of sherry or bitter ale, and bread.

If any fruit is taken, it must be either at breakfast or luncheon.

Dinner should be made of fresh, light fish, mutton, game, tripe, or underdone beef, floury potatoes, light vegetables, and a glass of sherry or claret; or, when it is indicated, a little brandy and soda-water.

Before retiring, a dry biscuit with milk and arrow-root, or a little brandy may be taken. All the food is to be thoroughly masticated, and eaten very slowly.

A few words as to what must be avoided in the diet of the aged.

Too much liquid at meals is injurious; for it dilutes the gastric juice, impedes digestion, and, if excessive, relaxes the stomach.

Tea, coffee, cocoa, and milk may be taken in moderate quantities with benefit, but are least objectionable an hour after meals.

Broths containing vegetables are extremely indigestible in the stomachs of the aged, and butter and cheese must be rare articles of diet.

Over-done, well-done meat, fresh bread, cabbage, carrots, turnips, veal, lamb, pork, salt meats, pastry and salads, are to be forbidden in the senile period of life. Remember Watson's saying: "*Gout* in the stomach should be oftener called *pork* in the stomach."

Shell-fish, lobsters and crabs, and salmon, herring, eels, mackerel and other kinds of rich, oily fish, must be avoided, for they often excite attacks of acute dyspepsia, whose gravity is out of all proportion, apparently, to the amount of the article taken.

Fermented liquors and oatmeal very frequently occasion flatulence and acidity of the stomach, so their use must be determined by the effects produced in each individual's case.

Variety is necessary, but it should never be at the expense of simplicity and wholesomeness of the food.

Next in importance to the diet, in the management of senile gastric catarrh, is the condition of the bowels.

The relief experienced after free alvine discharges is often so great, that the aged patient refers most of his troublesome symptoms to the constipated state of his bowels.

An aloetic pill may be taken regularly at dinner; and Hunjadi Janos or Friedrichshall are excellent laxative mineral waters.

Rhubarb and soda, or rhubarb and strychnia, are also efficient remedies for the chronic constipation attending senile gastric catarrh. The bowels must be kept regular, and hence the patient should go to stool at some stated time each day, whether desire is felt or not.

Exercise in the fresh air, cleanliness, early retiring and rising, and warm clothing, cannot be overrated as adjuvants to the treatment of catarrhal dyspepsia. A dry climate often produces markedly beneficial results.

Anorexia in old age is best treated with aromatic bitter infusions: quassia, columba, gentian and Peruvian bark, combined with strychnia, quinine and iron; and in the morbid sensibility of the stomach of old dram-drinkers, opium in small quantities will in many instances relieve the anorexia.

For the relief of the flatulence, the diet should be essentially *dry*. Vegetables should form but a very slight part of the food taken. Farinaceous food, potatoes especially, seems to disagree with this class of patients, while spices and the alkalies are often partaken of with benefit.

Cajeput oil, creosote, sulphite of soda, charcoal, and a combination of ammonia and the compound tincture of sulphuric ether, rarely fail to give at least temporary relief.

Cardialgia, or heartburn from acidity, is to be combated by the ad-

ministration of alkalies. Magnesia, potash and soda are much used, but lime-water in milk acts best, and gives more permanent relief.

Minute doses of morphine can be given when hyperacidity causes extreme irritation of the stomach; and bismuth with the bitter infusions also acts beneficially in allaying gastric irritability.

In pyrosis much benefit is often obtained by a change in the diet. Minute doses of morphia, with bismuth and bicarbonate of soda, in combination, may be given in extreme cases.

I have used kino, conium and belladonna with marked benefit in some cases.

For the relief of gastrodynia, nitrate or oxide of silver, with iron and rhubarb, has been highly recommended; but by far the most efficacious drug for its relief is the subnitrate of bismuth, which may be given in doses varying from five to twenty grains, three times a day, for an indefinite period. Hydrocyanic acid is a remedy that sometimes acts when all others fail, but it must be used with great care. When an attack of acute gastrodynia plainly arises from an overloaded stomach, an emetic of warm water may be given.

Hepatic, cardiac and pulmonary causes are to be treated according to the rules elsewhere given for their management; anæmia is to be combated with iron in the form of chalybeate waters.

If the gastric juice appears to be deficient in amount, pepsin and hydrochloric acid are to be given with the two principal meals of the day.

When, in old age, the dyspepsia is of nervous orgin, preparations of zinc, arsenic and phosphorus are useful; and a "little wine for the stomach's sake" often enables that organ to act more efficiently.

Diarrhœa in Old Age.

I shall briefly, in this connection, call your attention to a very frequent condition in advanced life, viz.: diarrhœa.

Just here it may be said that, when the trouble is of catarrhal origin, the pathological changes and morbid appearances differ in nowise from those of adult life; but a much milder and less extensive catarrh will induce serious symptoms than in middle life or childhood.

Etiology.—This condition is more frequent in those who grow old rapidly, and in many cases seems to arise from a state of the intestinal tract that is part of the senile marasmus; indeed, the name "*senile lientery*" has been given to one variety, so common is it at this period of life.

Epidemics of diarrhœa are not infrequently met with when large numbers of old people are crowded together in asylums and hospitals.

Errors in diet are prominent causes of senile diarrhœa.

Exposure to cold, sudden chilling of the body, and abruptly checking the perspiration are common causes of diarrhœa in the aged.

Aged gouty subjects have frequently a peculiar form of diarrhœa, which is intermittent and seems to be a sort of safety-valve, as great relief often follows slight attacks.

Diarrhœa is more prevalent in the winter than in the summer months.

I do not speak of the diarrhœa which, in the senile period of life, so frequently accompanies hepatic, renal, thoracic and various other chronic affections, and to which the name of symptomatic diarrhœa has not improperly been given.

Symptoms.—"*Senile lientery*" is the commonest form of diarrhœa in the

aged, and its most usual cause is over-feeding. You will very often find that many perfectly healthy old people have from four to six movements of the bowels each day, and that this condition has not only persisted for a long time, but it is also unaccompanied by any unpleasant symptom; indeed, some continue to grow stouter while the diarrhœa continues.

In this habitually relaxed state of the bowels, the stools are always pultaceous.

This feculent diarrhœa is most marked in those corpulent and sedentary old people whose appetite is undiminished with age, while their stomach and intestines are incapable of their former vigorous action.

When a diarrhœa coming on in old age is marked by limpid or brownish liquid discharges, it is called *serous;* it may be serous diarrhœa from the very onset, or it may be the result of neglect or improperly treated senile lientery.

In serous diarrhœa the normal fæces are first swept out by the early discharges, after which the motions consist of thin, ochry fluid, always offensive, and often frothy.

You will find that this form of diarrhœa almost always is the result of exposure to cold and wet; and at the commencement there may be some slight febrile movement, attended with rigors.

When partial or complete retention of the contents of the lower bowel accompanies a serous flux, the motions are scybalous, and the efforts to expel these hardened masses are constant and painful.

Catarrhal diarrhœa, or the mucous flux, differ in no way from the intestinal catarrh I have elsewhere spoken of, and is quite rare in old age.

Bilious dejections are usually attended with considerable exhaustion in old age. The tongue becomes furred, the complexion sallow; there is headache, and there may be a feeling of weight and uneasiness in the right hypochondrium.

When they continue even for a short time, the face becomes drawn and haggard, the eyes seem sunken, the pulse is weak and compressible; the temperature falls sometimes below the normal standard, and the patient dies in a short time from exhaustion.

It is to be borne in mind that, in advanced life, an acute diarrhœa may steadily persist while the patient utters no complaint, shows no signs of distress, and still the disease may be rapidly tending to a fatal issue. Griping and tenesmus are symptoms which are much oftener absent than present in senile diarrhœa.

Differential diagnosis.—This condition is not likely to be confounded with any other; but a diarrhœa may turn you away from a dangerous causative state, such as cancerous disease of the rectum or fecal accumulations in the rectum and colon.

A digital examination of the rectum will generally determine at once the latter condition.

Prognosis.—Diarrhœa is never without danger in old age. The outlook is worse the longer it continues, the more obstinate it is, and the graver its cause, as, for example, it is more serious when associated with cancer or ulceration of the rectum, than when it depends upon simple impaction or an indiscretion in diet.

Old age diarrhœa is frequently an attendant of hemorrhoids, and one of its commonest sequelæ is constipation. Death is caused by exhaustion.

Treatment.—When an old person has been over-indulgent at table, the plainest indication is to rid the intestine of the offensive material, and therefore such a laxative as castor-oil is to be employed.

Saline purgatives should be avoided, for in advanced life great exhaustion is frequently the result of a few rapidly induced watery evacuations. Hence it is always advisable to combine rhubarb, or its compound tincture, with the drug we administer; and if there is flatulence, half a dozen drops of laudanum may be advantageously added. In weak, aged patients, rhubarb, magnesia, and opium will be found to act remarkably well.

When the patient gives a history of a diarrhœa that has continued for some time, so that the bowels may be safely regarded as freely emptied, Dover's powder, combined with some such astringent as krameria or catechu, will be found to check it with great promptness. Chalk-mixture, combined with chloric ether and tincture of opium, is also valuable in similar instances.

When there is a marked febrile movement, Dover's powder is of the greatest value, and, in cases where prostration is not excessive, it may be combined with mercury and chalk.

When the case remains obstinate, and all these remedies have been tried, then you must resort to kino, catechu, logwood, tannic and gallic acids—the vegetable astringents—combined with opium.

Finally, the metallic astringents, copper, lead, silver, and iron, may be cautiously employed.

When senile diarrhœa is associated with a condition of anæmia, the mistura ferri compositus is of great utility, but, perhaps, the best combination under such circumstances is that used in those English hospitals where the inmates are mostly far advanced in life, namely, half a grain of cupric sulphate and a grain of opium twice a day, gradually increasing the dose of the former to three grains. The most rebellious forms succumb to this treatment.

When the urgent symptoms have passed, the dilute mineral acids, nitric and sulphuric, act beneficially, by first checking fermentative action, and then toning up the relaxed mucous membrane.

Suppositories are to be employed when there is much tenesmus, and when either an enema is inadmissible, or it is impossible to administer medicine by the mouth. Should there be any palpable cause which can be removed, the primary indication is, of course, to remove it.

Warmth is very efficacious, and often aids materially in checking a simple flux. An old writer, Wainwright, said: "A woollen shirt mightily conduces to cure an habitual diarrhœa." Warm flannels should be continually replaced over the abdomen; and in severe cases recourse may be had to warm poultices or hot fomentations.

The food should be of the most non-irritating character; alimentation must be chiefly amylaceous; and sago, rice, arrow-root, etc., in milk, are best borne. In some cases, wine with this diet seems to aid in checking the diarrhœa; and if the aged patient have been accustomed to wine for a number of years, it is not advisable to stop its administration.

Other stimulants must be avoided, as well as rich and highly seasoned food. It is best that, for a time, all meats should be avoided. It is imperative that an old person with any form of diarrhœa should have absolute rest in bed, and should avoid any sudden movement or mental excitement.

The recumbent posture prevents gravitation, and hence the rapid passage of the fluids over the irritated membrane.

A dry, stimulating atmosphere is the best for aged patients who suffer from diarrhœa; and if the diarrhœa can clearly be ascribed to a malarial cause, large doses of quinine are indicated.

When the principal pathological changes are in the rectum, much benefit

will be derived from starch enemata combined with opium or biborate of soda; rest and warmth, a simple diet, and a dry, bracing air will oftentimes effect what medicine cannot. Hence, hygienic treatment is of the first importance in senile lientery.

Next to the diarrhœa of old age comes properly the consideration of constipation.

Constipation in Old Age.

Habitual constipation is far more frequent in the aged than in adult life.

Etiology.—In most instances habitual constipation in the aged arises from loss of power to propel the contents of the intestine onward, and also from a diminution in the sensibility of the lower bowel. It may be caused by diseases of the brain and spinal cord; senile dementia, atrophy, softening, and hemiplegia are always accompanied by constipation.

The abuse of purgatives leads to chronic constipation, and constipation is often one of the results of long-continued senile lientery. The use of opium favors constipation. Collections of impacted fæces in the rectum have sometimes been found to surround masses of pills and other substances, such as magnesia, which were given to relieve constipation.

Change of diet, scene, or habit, anything interfering with the regular act of defecation, may cause temporary constipation, and this, in old age, is very apt to become habitual. Diminished contractile power of the abdominal muscles, the result of excessive development of fat, is a frequent cause of constipation in old age.

It may also result from unnatural dryness of the fæces, such, for example, as occurs in diabetes, where a very large quantity of fluid is carried off by the kidneys.

Torpor of the rectum, which may either be primary or follow prolonged inactivity of the upper portions of the large intestine, is a cause peculiar to the constipation of old age; and it is rare not to find it in feeble, infirm, bed-ridden patients, women especially; indeed, women suffer much oftener from this condition than men.

Constipation occurs more often in the sedentary and sluggish, where the calls of nature are neglected or postponed, in those who allow large fecal masses to accumulate in the intestine, and thus diminish its sensibility. In such case the constipation may be purely the result of habit, and hence have no other assignable cause.

Symptoms.—The aged submit to temporary constipation with indifference, a blunted sensibility of the intestinal canal favoring this. With the listlessness of advancing years, the dangerous habit of deferring to answer the call of nature leads to habitual constipation.

What may be regarded as constipation in one person is regularity in another. Thus, cases are on record where, from boyhood until the seventieth year, the bowels did not move more than once a week, and yet the individual enjoyed excellent health. Such individuals do not bear purging, and it is not safe to administer drastic cathartics to them.

In those accustomed to take large quantities of opium, the bowels have been known to move only four times in the year. The history of each individual will tell you if the bowels are sluggish, and whether the case is to be regarded as one of constipation.

When, in an old person with regular habits, two or three days pass without defecation, there is a sense of local fulness and heat, a tendency

to piles and flatulence, headache, vertigo, foul breath, anorexia, and well-marked dyspeptic symptoms. The patient is frequently hypochondriacal. If a predisposition to apoplexy exists, obstinate constipation often hastens the apoplectic stroke.

If a condition of constipation persists, vertigo becomes frequent, there are muscæ volitantes, tinnitus aurium, spasms of dyspnœa, sleeplessness, and occasionally a dull, steady, frontal headache ; the skin becomes parched, shrivelled, and sallow, and is liable to various kinds of eruptions, especially psoriasis, eczema, prurigo, erythema, and erysipelas. Injuries heal very slowly. The tongue is flaccid, and often indented by the teeth. The kidney and liver secrete morbid products, or the excrementitious bile being retained in the blood, it is rendered more or less impure. The ilio-hypogastric and ilio-inguinal nerves may be pressed on by the distended cæcum and adjoining colon, and neuralgic pains in the groin or over the iliac crest may result.

Hemorrhoids and flatulence are now marked ; there is more or less uneasiness in the genito-urinary tract because of the pressure from the tumor of indurated fæces, which likewise cause the piles and flatus. The veins of the lower extremity, testicle or ovary, may be pressed upon, and œdema of the feet and varicose veins occur. In prolonged cases the action of the stomach is crippled, and in many cases a cachexia is developed.

I shall continue this subject at my next lecture.

LECTURE XXX.

SENILE CONSTIPATION.

Summary.—Constipation in Old Age (*continued*)—Symptoms—Differential Diagnosis—Prognosis—Treatment.

Changes in the Bladder and Urine in Old Age—Atony or Paralysis of the Bladder—Definition—Etiology—Symptoms—Differential Diagnosis—Prognosis—Treatment—Chronic Enlargement—Hypertrophy of the Prostate Gland—Morbid Anatomy—Etiology.

GENTLEMEN:—Constipation is felt most from its *results*. Dilatation and hypertrophy of the intestine are very common conditions in old age. The dilatation begins just above the rectum, extends upward the entire length of the large intestine, the circumference in the latter intestine may measure from *nine to twelve* inches. Hypertrophy of the intestinal walls usually attends this dilation, and is best marked in the rectum and sigmoid flexure.

Pouches may form in the colon; and an elongated sac filled with mucus or fæces forms a sort of hernial protrusion as the circular muscular fibres yield. These pouches are most common at the sigmoid flexure.

Ulcerations may arise from prolonged constipation; perforation of the weakened wall and extravasation of the contents may lead to fatal peritonitis. Typhlitis and perityphlitis may arise from it. The colon and sigmoid flexure are altered in their position as well as in shape, their curvatures becoming more sharp than normal.

Disease of the rectum and contiguous structures, hæmaturia melæna, abscesses about the rectum, fistula, anal fissures, stricture, prolapse, varices of the prostate gland and the bladder, passive hyperæmia of the pelvic viscera, all are frequently met with in advanced life, and are in most instances clearly assignable to fecal accumulations, the result of habitual torpor of the bowels.

With torpor of the rectum, there is sometimes a spurious diarrhœa, accompanied by acute pain in the lower part of the abdomen, by great tenesmus and "bearing down" during defecation, and by the presence of scybalæ in the fæces. It may be accompanied by dysuria and even retention. Ileus and strangulated hernia have resulted from neglect of this torpor, which may be so complete that the relaxed and distended rectum, filled with hard fæces, occupies nearly the whole pelvic cavity.

The accumulated fæces frequently form tumors at different points along the digestive tract, and in emaciated subjects are easily detected on palpation. They are often felt in the transverse or ascending colon as movable masses; but the largest accumulations collect in the sigmoid flexure and cæcum, where manual palpation in the left or right ileo-inguinal region readily discovers their size and sometimes their consistence.

Differential diagnosis.—The differential diagnosis between spurious diarrhœa (torpor of the rectum) and senile lientery I have already considered.

Prognosis.—You will have observed that, in the consideration of senile

constipation, all malignant growths, all mechanical interferences other than fecal, have been disregarded; for when such are the causes, constipation is only a secondary symptom of a graver condition.

Hence, if we regard constipation as a retention of fæces in the lower part of the large intestine, the prognosis is very favorable, provided we can overcome the apathy and habitual indolence of the aged patient.

Inflammatory complications are very grave; fortunately, however, they are rare in the aged. Peritonitis, if it occurs, is always fatal.

Treatment.—An old person should go to the water-closet at the same time every day, whether he has the inclination or not. The slightest call of nature must never be disregarded.

Friction over the abdomen, or when the aged patient is confined to a chair, or is much debilitated, bending the body backward and forward will be found to provoke and aid defecation.

The galvanic current over the abdomen, along the course of the large intestine, and to the anus, acts beneficially in many cases—the *rationale* of its action being the same as that of friction, rubbing, and kneading of the bowels.

The diet should consist largely of vegetables, unless they are contraindicated. Prunes and figs are excellent gentle laxatives in the aged.

Oatmeal, when it does not excite flatulence and heartburn, may be taken every morning with molasses instead of milk. A goblet of fresh cold water just before retiring or on rising will, in many cases, relieve a sluggish condition of the bowels, and a cigar or pipe after meals often has the same effect. But in many cases vegetables, oatmeal, fruit, smoking, all disagree with the patient, or are not efficacious, and then recourse must be had to medicinal agents.

Many old people have taken first gentle, then stronger, and finally the most powerful aperients, with the belief that the bowels must move *every* day. In such cases you should call diet and exercise to your aid, and return to the milder laxatives, endeavoring to produce a healthy evacuation every other day. *Tonics* should always be combined with the laxatives; indeed, many have gone so far as to regard strychnia, iron, or quinine, when given alone, as able to effect a radical cure in most cases.

Colocynth, gentian, and quinine, is an excellent combination for a pill in mild cases. So also is a pill composed of aloes, rhubarb, and strychnia, or iron. Another efficient combination is aloes, myrrh, and gentian, combined with quinine, strychnia, or iron.

Podophyllin often produces slow and painless evacuations, and deserves a careful trial in the treatment of senile constipation.

In very obstinate cases the treatment may begin with the exhibition of the compound extract of colocynth, scammony, and about the sixth of a drop of croton-oil. One or two of such pills may be taken before dinner or at bedtime.

These may be continued for a long time. Maclachlan records a case of a lady one hundred and three years old, who for the last fifty years of her life took a compound aloetic or rhubarb pill at bedtime. Brodie speaks of a gentleman, eighty-six years of age, "who for three-score years took an aloetic pill every night." The general rule in advanced life should be to use only mild laxatives, and to change them frequently; the continued use of drastic purges in old age is apt to lead to trouble.

When colonic or cæcal accumulations occur, active purgation is necessary; at the same time the aged patient must be sustained by stimulants and nutritious food.

When the lower part of the bowel is clogged, the mass will often have to be scooped out; and, when very hard, this operation is best preceded by a steady stream of moderately hot water against the mass for about half an hour.

Purgative enemata should be adjuvants to all forms of treatment, looking to the expulsion of a large mass that has been accumulating for a long period.

A case is recorded in the "Cyclopædia of Practical Medicine," of an old gentleman—paraplegic—who had a gallon of salt water thrown up every morning for a week after constipation of long duration. After several days an enormous mass of fæces passed, the activity of the muscular fibres having finally been called into play.

I shall now briefly invite your attention to diseases of the bladder; as we meet them in the aged, they are very different from those of adult life.

Atony or Paralysis of the Bladder.

Vesical paresis is frequently met with in the aged of both sexes; it is essentially a condition of advanced age.

In accordance with the plan I have adopted, a few words are necessary in regard to the changes which are normal to old age, and, at the same time, different from those of adult life.

Healthy old people secrete less urine than in middle life, and the color of the fluid is paler, less rich in solid constituents, and of lower specific gravity than normal adult urine. Epithelium from the various portions of the genito-urinary tract and mucus are usually quite abundantly mingled with it.

The amount of urea secreted by very old men is small—only one hundred and twenty-five grains in twenty-four hours, being noticeably less than the amount in the urine of young children. The amount of uric acid is also diminished by about one-half.

The quantity of urine discharged in old age averages sometimes as low as fifteen or twenty ounces per diem, and this is compatible with perfect (senile) good health; but, when not more than six or eight are voided, the system soon indicates the presence of an abnormal quantity of urea.

By atony or paresis of the bladder is understood a lack of power in its muscular coat, by which its longitudinal and oblique muscular fibres and the detrusor urinæ do not contract, or only do so in a very tardy and imperfect manner.

Etiology.—In a limited sense, atony of the bladder is physiological in old age; it is well known that the distance to which a boy can throw a stream of urine from his bladder is greater than that of an older person, and so the power of expelling the secretion goes on diminishing with advancing years.

There are two reasons for this: one is, that there is a steady loss of muscular power, a senile decay throughout the entire muscular system, the muscles losing their normal contractility; and the other is, that the older the person, the more engaged in business cares he will be, and hence, the calls of nature to void the bladder are very frequently and habitually unanswered. Thus, there is a certain degree of habitual distention of the bladder, not only during the day, but at night as well.

This disregard to empty the bladder may be, as I have just said, volun-

tary. Involuntary neglect occurs in prolonged alcoholic intoxication, and in all those diseases, whether acute or chronic, which affect the sensorium —among these may be mentioned small-pox, typhoid, typhus, paraplegia, apoplexy, epilepsy, and diseases of the spinal cord.

Paralytic weakness of the bladder in old men is very frequently secondary to an enlarged prostate; and it *may* result, in either sex, from the individual being compelled to remain in a recumbent position a long time.

Senile catarrh of the bladder may lead to subsequent paresis, from implication of the muscular coats in the process.

Women who have had large families, and whose labors have been severe, are especially prone to palsy of the neck of the bladder; the obese suffer oftener than those of the opposite condition.

Paresis of the bladder, though common to both sexes, is more frequent in men from prostatic enlargement; there seems, too, to be a predisposition in some to atony of the bladder, and in them you will find such causes as passing from a heated room to the open air, or the slightest over-indulgence at table, sufficient to induce temporary attacks of retention of urine.

Finally the more impaired the general health, and the more sedentary the life of the individual, the greater the predisposition to atony of the bladder.

Symptoms.—The bladder, unlike the rectum, retains its contents when paralyzed; for the elasticity of the sphincter vesicæ is inherent in the tissue itself. Thus retention occurs; or, if the sphincter be involved also, we have a combination of incontinence and retention; paradoxical as it may appear, only a part of the urine passes off, and that by its own gravity, aided, perhaps, somewhat by the action of the abdominal muscles, after a considerable quantity has accumulated in the bladder.

The aged patient notices that there is a gradual diminution in the power of emptying the bladder; that, after he wills to micturate, quite a time elapses before the first drops come, and that the act is more prolonged than usual, the urine falling perpendicularly and only becoming a full stream at about the middle of the act.

The desire to micturate is now less often felt; and if, when felt, it is disregarded, the desire soon disappears, as if the bladder had been emptied.

The stream is feeble and interrupted; occasionally the urine flows away either in drops or interruptedly, and the odor is usually disagreeable on account of its ammoniacal decomposition.

This latter condition is generally the accompaniment of a paralyzed sphincter with vesical atony.

After the patient thinks the act of urination is complete, a few drops will suddenly pass; not infrequently those in advanced life have an escape of urine during the night.

When the bladder has once become greatly distended, a considerable quantity of urine continues to dribble from it, the *retention* being still unrelieved.

This *retention with dribbling* in old persons is a condition the true nature of which is frequently unrecognized. Cases have arisen where supposed tumors have been tapped, and a distended bladder entered, and a large amount of retained urine drawn off which was never suspected.

In the very old the urine may steal away from the bladder as it flows drop by drop from the ureters.

When retention is excessive the bladder slowly enlarges, finally rising up into the abdomen even as high as the umbilicus.

On *palpation*, the distended bladder is felt round or pyriform in shape, hard, and elastic, projecting above the pubis; over it you may detect fluctuation through the abdominal walls.

Percussion elicits well-marked dulness, varying in extent with the amount of distention.

If a condition which gives these physical signs is not relieved by the catheter, the aged patient will either have true incontinence following, the urine flowing away uninterruptedly; or there may develop uræmia, ammonæmia, intense catarrh of the bladder, or even vesical gangrene.

In all cases the true condition of the bladder is readily determined by the introduction of the catheter; and, in those advanced in life, and especially when occupying a recumbent position, it may be necessary to press the hand over the bladder in order to expel the urine.

Differential diagnosis.—Atony of the bladder in old age may be confounded with retention from obstruction, and with ascites.

In *atony* the urine will flow out slowly, or in faintly marked jets, in obedience to the respiratory acts, when the catheter is introduced, oftentimes pressure from without being necessary to expel it. In retention from obstruction, the introduction of the catheter is attended with more or less difficulty at some point; but, having once entered the bladder, a free stream is projected in a single jet.

Ascites has misled many from atonic retention, and some have tapped the abdomen for dropsy. The introduction of the catheter is an unfailing means of diagnosis.

Prognosis.—The results of not attending to atonic retention are various and grave.

The constituents of the retained urine becoming absorbed, and the skin thus being charged with the extra duty of elimination of part of them, chronic eczema or some other form of intractable cutaneous affection may occur, only to yield when the bladder is freed from all *residual* urine.

Vesical catarrh, ammonæmia, uræmia, and gangrene or sloughing of the bladder, are the complicating conditions which may ensue when the case has been of long standing.

Complete recovery rarely occurs in these cases. The worst cases are those where paralysis and over-distention of the bladder have existed a long time. In most of these cases, even when the bladder is regularly emptied, the patient is only relieved, not cured, the progressive loss of strength continues, and, finally, comparatively rapid dissolution results.

The prognosis becomes unfavorable when the patient has a rapid and feeble pulse, a furred tongue; when the appetite fails and the nights are restless; mental depression and final stupor supervenes.

Death commonly occurs, in such cases, from exhaustion or uræmia.

Finally, the prognosis is influenced in a great measure by the age of the patient and the extent and duration of the atony.

Treatment.—Old people cannot have it too thoroughly impressed upon them to empty the bladder directly the call of nature is felt. This is a most important prophylactic measure.

If the patient be an old invalid, he must assume a position on the knees when micturating, for otherwise the bladder is incompletely emptied.

Petit has recommended that in simple atony the patient should press the cold chamber firmly against the thighs and scrotum; or cold lotions, cold hip-baths and cold sponging may be resorted to.

The catheter must be passed three or four times daily, so as to protect the muscular fibres from any further distention. But do not practise ca-

theterization so often that little urine shall collect in the bladder, and in this way convert the organ into a mere passage. The patient should learn to pass the instrument himself.

If injections into the bladder are resorted to, the amount of fluid injections must not be more than four ounces at a time, and the temperature should be gradually lowered; thus, the first injection should have a temperature of about ninety-four or ninety-five degrees Fahr., the second ninety degrees, and the third and last eighty-five degrees Fahr. If vesical catarrh exist, never inject an old person's bladder with cold water.

Galvanic and electric currents are often of temporary service in senile atony of the bladder; the powerful internal remedies that have been tried and found to act beneficially in certain cases, are strychnia, cantharides, creosote, turpentine, and ergot.

The general health of the patient should be carefully attended to; for, without nutrition at a high standard, we cannot expect the muscular system, and this muscle as part of it, to regain, or at least to improve in its power.

Hence, in most cases, iron, strychnia, the vegetable tonics and quinine, form a very important part of the treatment; the bowels must be regulated with aloetic pills; indeed, some claim that direct and permanent benefit follows the use of aloes.

Balsams, which are highly recommended, derange the already weak digestion of old age, and vesicants and embrocations possess little or no advantage. The urine may be made alkaline in some cases to prevent cystitis, when this threatens.

Finally, if there be constant dribbling, a portable urinal should be affixed, and the most scrupulous cleanliness enjoined. Of all the preparations of iron, the muriated tincture is the most serviceable.

Closely connected with vesical paresis is

Chronic Enlargement, Hypertrophy of the Prostate Gland.

I use the term "*chronic*" enlargement to distinguish senile prostatic enlargement from that which may occur in adult life from inflammation.

Enlargement of the prostate gland is by some regarded as a *physiological* condition in old age, while others state that it is a diseased condition incident chiefly to, but not wholly caused by old age.

Morbid anatomy.—Prostatic hypertrophy may be general or partial, and is almost entirely confined to its muscular tissue, although the glandular substance *may* be slightly involved. Pathologically, this condition of the gland may be classed among *myomata* of the prostate; when the glandular structure is implicated the name *adeno-myoma* is applicable.

Cases are recorded where in very old men the gland had acquired the size of the two fists, or even that of a child's head, nearly filling the lower basin of the pelvis. But, when it reaches the size of an orange, it may be regarded as exceptionally large, as it is generally about the size of a hen's egg.

The color, externally, is unaltered. The so-called third lobe is frequently involved, and may acquire the size of a hickory-nut the remaining portion of the gland being unimplicated; this is called *median centric hypertrophy.* When lateral hypertrophy exists, it is rare to find one lobe affected to the exclusion of its fellow.

The tumor in general hypertrophy may be smooth, rounded, and regu-

lar, but it is rather the rule for unsymmetrical enlargements to be present, the gland presenting great irregularity of outline. Cases are recorded where it has weighed twenty ounces.

On *section* the cut surface pushes up above the level of the cut, and the alternations in color are more strikingly marked than in adult life. A viscid fluid, the thickened prostatic secretion, sometimes fills the acini and may be mistaken for pus. In many cases there is no fluid whatever in the gland.

In the centre of the mass you will frequently find numerous small, dense fibrous tumors, rarely exceeding the size of a pea. Prostatic concretions or calculi are also very frequent accompaniments of this condition, and are found in the interior of the glandular acini or ducts. These are round, colorless masses, varying in size from $\frac{1}{6000}$ of an inch to $\frac{1}{25}$ of an inch in diameter, and resembling colloid material in their action with tincture of iodine and sulphuric acid. Larger calculi are sometimes found in hypertrophied prostates, consisting of laminæ of oxalate or phosphate of lime, very difficult to crush.

In some instances, pedunculated hypertrophic prostates are found, which surround the neck of the bladder like a collar.

The veins of the gland are found dilated and tortuous.

The urethral canal is in nearly all cases more or less implicated. When lateral hypertrophy predominates, it is twisted; and when the middle lobe is chiefly enlarged, it is flattened and compressed.

The prostatic portion of the canal is always elongated and expanded, so that it may become capable of holding two or three ounces of urine. This elongation also carries the neck of the bladder upward and behind the pubes.

Etiology.—Old age is an essential condition for prostatic enlargement. Its exact etiological relationship with aortic insufficiency, or with chronic pulmonary affections, is not yet definitely determined.

The condition is rare before fifty, and quite an exceptional circumstance before sixty; subsequently it is of quite frequent occurrence.

Sir Benjamin Brodie used to say: "When the hair becomes gray and scanty, when specks of earthy matter begin to be deposited in the tunics of the arteries, and when a white zone is formed at the margin of the cornea, at this period the prostate gland usually, I might say, perhaps, invariably, becomes increased in size."

Sir Astley Cooper even went so far as to regard hypertrophy of the prostate as a salutary process, since "it prevents incontinence of urine, which in the aged would almost always take place were it not for this preventive."

Sedentary habits, over-indulgence in venery, the contrary state of affairs, gout, high living, hard riding (as in cavalrymen), all these have been assigned as causative conditions for an enlarged prostate, but no satisfactory proof exists to substantiate the assertions.

One peculiar point, which it may not be out of place to mention, is that organic stricture of the urethra and enlarged prostate are among the *rarest* coincidences of advanced life.

LECTURE XXXI.

SENILE HYPERTROPHY OF THE PROSTATE GLAND.

Summary.—Symptoms—Differential Diagnosis—Prognosis—Treatment. Ammonæmia—Definition—Morbid Anatomy—Etiology—Symptoms—Differential Diagnosis—Prognosis—Treatment.

GENTLEMEN:—At my last lecture I spoke of the etiology of senile enlargement of the prostate gland; we shall now turn to a consideration of its symptoms.

Symptoms.—Prostatic enlargement may exist for a long time to a limited extent without producing any symptoms that direct attention to the genito-urinary tract. A case is recorded where, at the autopsy of a man who died of old age at one hundred and one, the organs were all normal (at the senile standard), except the prostate, which was exceedingly hypertrophied, and no symptom of its presence existed during life.

If this condition induce a "bar at the neck of the bladder," there may be retention of urine; there is always in such a condition more or less residual urine in the bladder.

The increased mucous secretion from the hyperæmic part, mingling with the stagnant urine, aids greatly in its decomposition; and the carbonate of ammonia thus set free increases the inflammation and induces retention, and thus *distention of the bladder* is a constant result of an enlarged prostatic gland.

In the mildest form of chronic enlargement, the old man notices that he micturates oftener during the day than he used to, and that he has to rise a little earlier in the morning on account of a pressing desire to urinate. There is a sense of uneasiness, or actual pain of a stinging kind, extending along the penis, felt more especially at the glans.

He has to wait a little time before the flow begins, and more or less straining is always necessary to start it; then it comes slowly and cannot be projected in a jet; indeed, it usually falls nearly perpendicularly from the urethra. After he thinks the act complete, several drops pass; and in careless old men, especially in summer, this is more inconvenient to others than to themselves. The stream is always small.

After a time he is compelled to evacuate the bladder every hour or two, or there may be a continual dribbling, especially marked when the patient is in a recumbent posture.

There is no sense of satisfaction after micturition; and just here it may be mentioned that, when excessive prostatic enlargement exists, there is a sense of incomplete evacuation of the rectum, and tenesmus is occasionally present at stool. For the same reason, hemorrhoids and prolapsus ani are by no means infrequent coexisting conditions.

Any slight indiscretion in eating and drinking may bring on an attack

of retention. All violent or jolting exercise, such as horseback-riding, increases the desire to micturate, and may cause slight pain along the course of the urethra, together with flying pains in the hips, limbs, and about the pubis.

The irritation from an enlarged prostate often excites such lascivious desires in old men that they become notoriously indecent. Such cases are common everywhere; in every village there are one or two old men who are terrors to the maidens.

In the severer form, the bladder is exceedingly irritable; a sense of weight and fulness is experienced in the perinæum; retention soon follows, the other symptoms being those of the milder variety.

The efforts made to expel the urine are often so severe that various portions of the mucous membrane, weaker than the rest, are pressed out, and sacculation of the bladder is the result.

The urine decomposing in these sacs is one of the most favorable conditions for the formation of stone.

When the outflow has been obstructed for some time, structural changes occur in the muscular and mucous coats of the bladder; there is usually a mild or severe chronic catarrh of the ureters and calices and pelvis of the kidney.

Sometimes the bladder hypertrophies and contracts, instead of becoming distended; then irritability of the bladder becomes a marked and constant symptom.

As the disease progresses, and when the mucous membrane is inflamed and ulcerated, the urine scanty and containing blood and pus, the countenance becomes sallow and indicative of organic disease.

Finally, worn out by sleepless nights and continual pain, the aged sufferer sinks from exhaustion, retaining his faculties till the last; or he dies more rapidly in a coma which was preceded by typhoid symptoms—a dry, brown tongue, general prostration, rapid, feeble, and irregular pulse, and low, muttering delirium. The urine may be alkaline or acid.

The first changes in it are a fetid smell, and the presence in the secretion of viscid, stringy mucus. As the disease progresses, more and more of residual urine is left in the bladder, and we find it dark and mingled with gummy mucus. When vesical catarrh is present, the urine may have an almost milky appearance, from admixture of pus, and possess a horribly fetid and ammoniacal odor, blackening the silver catheter. It is rare to find the urine of an old man with enlarged prostate that does not contain pus-globules, blood-corpuscles, amorphous urates and phosphates, mingled with crystals of the triple phosphates and stringy mucus.

A *physical examination* per rectum discloses a rounded or nodulated, lobular, dense tumor, in the region of the prostate gland. Pressure upon this tumor will usually excite a desire to urinate.

Percussion may reveal an enlarged bladder.

The catheter cannot be easily introduced into the bladder, on account of the obstruction it meets from the enlarged gland. In the attempt to explore the urethra a gum-elastic catheter should be used; and if, in endeavoring to make a diagnosis by catheterization, there is much difficulty and *any* doubt, Squire's vertebrated catheter, or the soft, bulbous-pointed prostatic catheter, is probably preferable to any other instrument, as both can turn very abrupt curves. Besides, there are multitudes of instruments which it is unnecessary to enumerate for diagnosticating the amount and situation of the urethral twisting and narrowing which come from hypertrophy of the prostate.

Differential diagnosis.—Senile prostatic enlargement may be mistaken for atony of the bladder, stricture of the urethra, and stone in the bladder.

Atony is readily diagnosed from hypertrophy of the prostate by the manner in which the urine flows from a catheter. In enlargement of the prostate the flow is quite forcible, and can often be made more so by the patient's will; whereas in atony of the bladder the urine merely flows out from the instrument, and no augmentation can be voluntarily made in its force by the patient.

Stricture is not a disease of advanced life. In stricture there are no rectal evidences of a tumor on physical exploration, while these are present in hypertrophy of the prostate. In stricture there is usually a history of venereal disease, which may be absent in hypertrophy. A sense of incomplete evacuation of the bladder is present in cases of prostatic enlargement, and absent when organic stricture alone is present.

The urine in stricture is *normal*, while in enlargement of the prostate it has the abnormal ingredients which I have just described.

Again, the catheter meets an obstruction about *six* inches from the meatus in stricture; and in enlargement of the prostate the distance of the obstruction is at least *seven* inches from the meatus.

Stone is very difficult of recognition, as contradistinguished from a hypertrophied prostate.

When blood is passed after exercise, it indicates the presence of a stone, and in the latter condition the difficulty in micturition has been as long and as steadily coming on as in enlarged prostate.

The tumor in stone, if felt *per rectum*, is movable; while it is immovable in prostatic hypertrophy. The sound is the only means which we have of making a positive diagnosis.

Prognosis.—We cannot cure enlargement of the prostate in old age, nor can we check its progress when once it is developed; it is unquestionably a part of senile decay. Its duration, then, is indefinite. The outlook for comfort is best in those cases where there has never been complete retention of urine.

The effects of an enlarged prostate in the aged vary greatly, and necessarily modify the prognosis. A moderate enlargement of the "third" lobe, or of one or both of the lateral lobes is sometimes attended by retention of the urine and great difficulty in passing the catheter; while cases where the autopsy has revealed a prostate as large as a base-ball had caused during life no difficulty with the urine and no obstruction to the passage of a catheter.

This condition may become very grave from the complicating conditions which are rarely altogether absent during its course; these are cystitis, pericystitis, vesical ulceration, overflow, inflammation of the ureters, pyelitis, nephritis, perinephritis, enlargement of the testicle, hemorrhoids, prolapsus ani, and ammonæmia.

Treatment.—We have no means of arresting the slowly advancing chronic enlargement of the prostate gland in advancing life.

If anything will check its progress, it is a diet and mode of life which are the opposite to those which are known to favor rapid and extensive hypertrophy.

Alcohol, if used, must be used moderately, or it is better not to use it at all. The slightest call of nature to evacuate the bladder must be immediately obeyed. Horseback-riding is to be avoided. Flannels must be worn next the skin, the extremities especially being kept warm.

Ultimately the regular daily introduction of *the catheter* will become a necessity.

As a rule, old men are very much opposed to the introduction of an instrument into their bladders, and it may require much persuasion and a clear description of the exact state of affairs before they will permit it.

Again, it is to be remembered that catheterism is sometimes dangerous in old men; the following statements will tell you at once not only to postpone their use as long as possible, but it will also indicate to you the importance of gentleness and care in the manipulation:

An old man, eighty, has been troubled with some difficulty in passing water. A bougie is passed carefully till it reaches the prostate, when it stops. It is then withdrawn, for no force is used to push it farther, and a little blood follows its removal. After that day *not a drop* of urine passes except through the catheter.

Another case resembles this precisely, except that, following the few drops of blood, there is complete retention, which results in death in a few days.

The shock from a cold sound passing over the prostate, even when slightly hypertrophied, may give rise to urgent symptoms, and *has* caused death.

Still a catheter must be introduced, and when introduced should be of the temperature of the body, the patient always standing during the operation.

The bladder is to be emptied at least twice a day; and if there is cystitis, glycerine or biborate of soda may be added to the water used to wash out the viscus.

Alkalies, administered with flaxseed tea, are very good internal remedies when the urine is strongly acid.

During warm weather, old men may not require the catheter; but, as soon as cold weather approaches, it will be necessary to resort to it again.

Retention and catarrh of the bladder demand prompt and appropriate treatment, for in old age they rapidly bring on a condition which may end in death.

Ammonæmia.

In connection with enlarged prostate and atony of the bladder, comes the consideration of a disease which, though rare during adult life, is frequently met with in those advanced in life.

Ammonæmia is that change in the blood due to the presence of carbonate of ammonia, which arises from metamorphosis of urea, the result of *retention* of the urine in the urinary organs.

Thus it is evident why ammonæmia is frequent in old age, for in that period of life atony of the bladder and enlarged prostate are frequent conditions.

Morbid anatomy.—The urinary tract will present the appearance of more or less acute catarrh, as well as conditions favoring or causing urinary accumulations.

There is always chronic catarrh in the intestines, and it has been observed that they were filled with a greenish yellow mucus and an alkaline fluid, having an ammoniacal odor.

Ulcers have also been found in the large intestine, similar to those in *dysentery.*

It may be mentioned here that in the cases where ammonæmia occurs with uræmia, which is quite rare in the aged, it is possible for the urea excreted into the intestines to change into carbonate of ammonia, and thus bring about the pathological condition I have just described as sometimes existing.

Rosenstein denies any connection between ammonæmia and poisoning by carbonate of ammonia, but the weight of opinion is against his statement.

Etiology.—All that is required to produce ammonæmia is the retention of urine in the body sufficiently long to allow of the metamorphosis of its urea.

Decomposition occurs very quickly, and of course, more rapidly in, than out of the organism, on account of the bodily warmth.

Ammonæmia occurs with enlarged prostate, atony, and paralysis of the bladder. These are the chief causes in old age, although it may arise at that period from stricture of the urethra, sacculated kidney, pyonephrosis, and hydronephrosis.

Symptoms.—Ammonæmia may be divided into two forms, according as the inducing cause is of sudden but permanent occurrence, or comes on gradually and steadily.

In the first, the so-called "*acute*" form, there are nausea and vomiting, intermittent chills, acceleration of the pulse-rate, followed by a rise in temperature. Diarrhœa is also a frequent accompaniment of acute ammonæmia.

The complexion rapidly becomes dingy and bronzed; and there is great muscular weakness, with a tendency to lethargy and stupor.

Rarely, however, are there any convulsions or œdema of the feet.

The tongue is brown, dry, and shining—the "beefy tongue;" the mucous membranes are remarkably dry, that of the throat especially; and the perspiration and breath have a well-marked ammoniacal odor.

In the "*chronic*" form, that which comes on in old men with enlarged prostates or atonied bladders, the complexion gradually passes from a sallow to a dingy brown hue, and there is slow, but progressive emaciation.

The aged patient is restless, has a slight headache, and insomnia becomes a very distressing symptom. Now and then chills occur, but with no regularity, and vomiting is an important symptom. Meanwhile, as the complexion darkens and emaciation progresses, the mucous membranes begin to assume a dry, glazed, shining look, the skin becomes drier and drier, and the breath and perspiration take on a distinctly ammoniacal smell, but the amount of perspiration is greatly diminished.

The temperature in these prolonged cases is constantly above the normal; and as the condition gets worse, the pulse is each day more and more accelerated.

With these symptoms there will be no œdema of the feet, or, in the majority of cases, convulsions; but the symptoms will rather counterfeit, to a very striking degree, those of chronic gastric catarrh. Persistent vomiting is often a prominent symptom. The bowels are usually constipated, although at times slight attacks of diarrhœa alternate with the constipation.

Finally, after emaciation has become extreme, and a cachexia has been fully developed, the restlessness and insomnia give way to lethargy, stupor, and the patient passes into a typhoid condition.

In old men with enlarged prostates this is quite a common termination,

the patient passing from stupor into the comatose state, with low, muttering delirium, rapid, feeble, and irregular pulse, and finally dying in a condition of deep coma.

The URINE is ammoniacal, and hence strongly alkaline *when passed*, frequently containing pus and depositing amorphous phosphate of lime with crystals of ammonio-magnesia phosphate. Its odor is as offensive as it is pungent.

Differential diagnosis.—Senile ammonæmia may be confounded with "typhoid gastritis," pyæmia, and septicæmia.

In *typhoid gastritis* the urinary symptoms are negative, in ammonæmia they are diagnostic.

In gastric catarrh there is no particular odor to the breath or perspiration, while this is markedly ammoniacal in cases of ammoniacal poisoning.

Nausea and vomiting may be prominent symptoms in chronic ammonæmia (the only form which would lead to confusion), while they are very slight or absent in gastric catarrh.

In the ammonæmia of the aged, the catheter or a rectal examination will show the existence usually of atony of the bladder, or hypertrophy of the prostate, while you will find no genito-urinary causes in typhoid gastritis.

Pyæmia and *septicæmia* may be mistaken for ammonæmia, on account of the color of the skin and lethargic expression of the face.

In *pyæmia* there is the history of a severe *initiatory* chill, profuse recurring sweats, high temperature—102° to 104°—sweet, sicky breath, and an appearance of infarctions, thrombi, or multiple abscesses in some organ. None of these symptoms occur in ammonæmia; on the contrary, the odor of the breath, the condition of the urine, and the evidence of some mechanical obstruction, would all be present, and with the other points would suffice to establish the diagnosis.

In *septicæmia* the temperature is much higher (105°—107°) from the onset; there are no recurring chills, no ammoniacal odor to the breath or body, and no urinary evidences such as are found with ammonæmia.

And finally, in both pyæmia and septicæmia the skin is in a strikingly opposite state to that of ammonæmia, being for the most of the time bathed in a copious perspiration, while in the latter disease it is dry, harsh, and has an ammoniacal odor.

Prognosis.—The prognosis in ammonæmia is determined to a great extent by the conditions which cause it.

When their removal is possible—as in retention of urine from enlarged prostate—the prognosis is favorable.

When it is due to pyelitis or sacculated kidneys, it is very bad; in all cases there is a gradual and steady impoverishment of the general health; old people suffering from any blood-poison are liable to sink rapidly into a typhoid state if the conditions which give rise to the poisoning cannot be speedily removed.

Treatment.—Its treatment consists in removing its cause. This is possible in a large proportion of cases, since atony of the bladder and hypertrophied prostate are by far the most frequent conditions.

Very often, when the aged patient seems fatally sinking, and when no suspicions have been attached to the bladder, you may draw off a large quantity of stinking urine, and then, with subsequent washing of the viscus, a rapid improvement takes place, and the gastric symptoms subside.

The diet of this class of patients should always be supporting and stimulating.

Atony of the bladder and hypertrophy of the prostate must be treated

according to the rules already given, and the catheter will here be most useful, and should be passed at least twice every day.

To the tepid water used to wash out the bladder, you may add, at discretion, carbolic acid, biborate of soda, or glycerine.

The washings should be continued until the withdrawn fluid is perfectly clear and inoffensive in odor.

The treatment of this condition, beyond the removal of the cause, resolves itself into supporting and nourishing the patient, and relieving those catarrhal inflammations which have been excited by the ammoniacal poisoning.

DESCRIPTION OF PLATES.

PLATE I.

FIG. 1.—Right hand of a man sixty-nine years old, attacked with gout since his thirty-third year. A large tophus is seen at the base of the index-finger, on a level with the metacarpo-phalangeal articulation. A second tophus, smaller than the preceding, is situated at the base of the middle finger. There are numerous concretions of urate of soda upon the external ear of this man.

FIG. 2.—Left hand of a gouty woman, eighty-four years old, who died in the *Salpêtrière*, in 1863. Both hands were symmetrically affected, and to the same degree; there was no appearance of tophaceous concretions in the vicinity of the joints. Here we find an exact reproduction of one of the types of deformities of the upper extremities most frequently seen in progressive chronic articular rheumatism. The articular cartilages of the metacarpo-phalangeal articulation were encrusted with urate of soda. Upon the dorsal aspect of the metacarpal heads there were, besides, tophaceous deposits which, situated immediately beneath the skin, and pressed against the heads of the bones, were flattened, forming no appreciable projection upon the back of the hand; this occurred in such a way that, before dissection, their existence could not be recognized.

FIGS. 3, 4, 5, and 6.—These refer to the anatomy of *Heberden's nodes*. In Fig. 3 the second phalangeal articulation is seen deformed, but still covered by the soft parts. The pisiform projections described by Heberden are well marked.

FIG. 4 shows the ends of the bones laid bare by dissection; the articular surfaces are broadened in all directions and thickened, on account of the formation of osteophytes.

FIG. 5.—The same preparation viewed laterally.

FIG. 6.—Normal condition, with which Fig. 4 is to be compared.

Fig. 1.

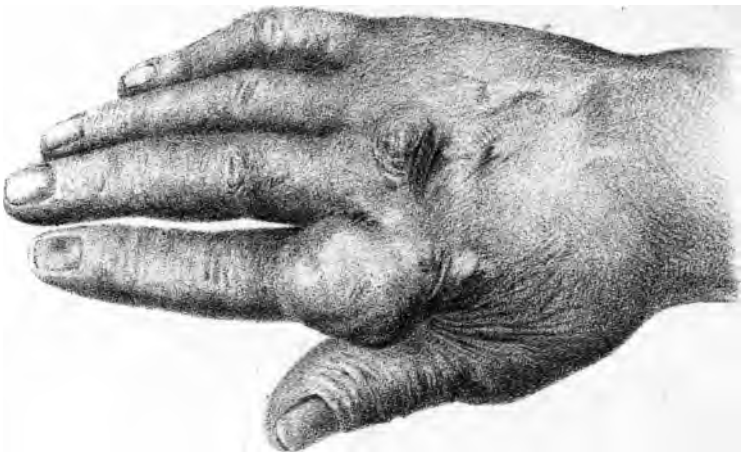


Fig. 2.

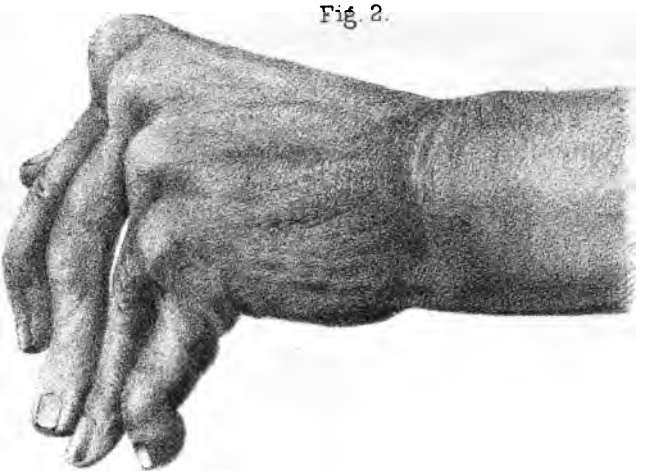


Fig. 3.

Fig. 4.

Fig. 5.

Fig. 6.

PLATE II.

FIGS. 1 and 2.—Deformity of the hands in general chronic articular rheumatism. The characteristics of the *first* type are well shown in Fig. 2. Fig. 1 gives a good idea of the deformities occurring in the *second* type.

FIG. 3.—Changes in the mitral valve in a case of primitive general chronic articular rheumatism.

Fig 1.

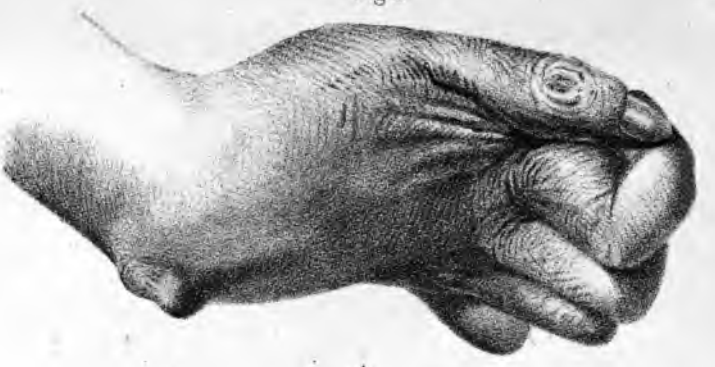


Fig 2.

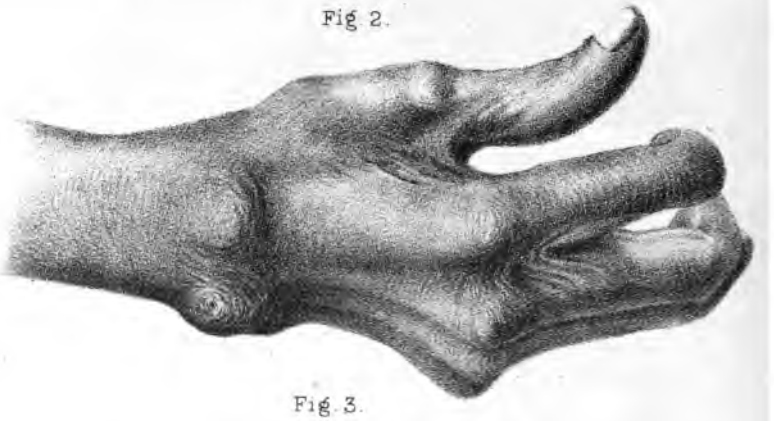


Fig. 3.

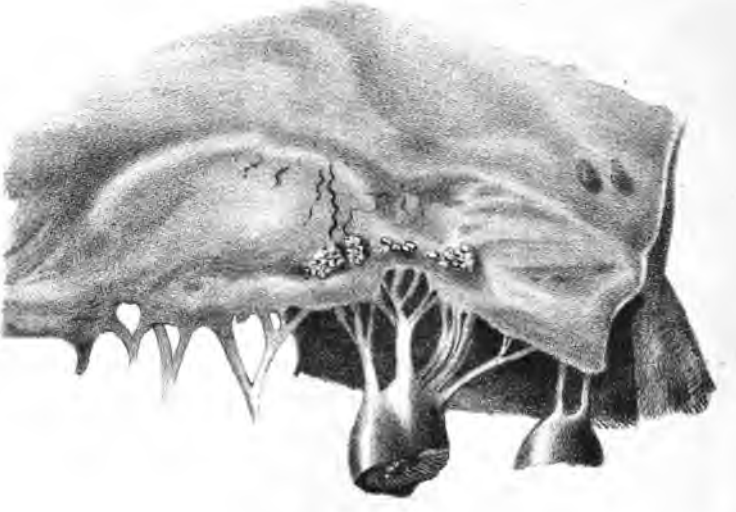


PLATE III.

FIG. 1.—*Gouty nephritis.*—Part of a section of the kidney (10 diameters); the white, chalky-looking lines (*a*) are deposits of urate of soda occupying the medullary (tubular) substance. These are represented in Fig. 3, magnified 150 diameters.

FIG. 2.—A convoluted uriniferous tubule of the cortical substance, whose epithelial cells (*b*), large and clouded, are also filled with fatty granulations. (300 diameters.)

FIG. 3.—Crystals of urate of soda (*d*), forming the deposit in Fig. 1 (*a*), visible to the naked eye. (Section of the tubular portion, 150 diameters.)

FIG. 4.—This figure has reference to the period of dissolution of these deposits under the influence of acetic acid. The free crystals are dissolved, and nothing remains but an amorphous deposit (*e*), which slowly goes on dissolving. It is then very clearly seen that a portion of this deposit is situated in the interior of the uriniferous tubules (*g*). (Section of kidney, 200 diameters.)

FIG. 5.—Synovial fringes from the knee-joint covered with their epithelium, and exhibiting at (*m*) a deposit of urate of soda, generally amorphous. (N. B. —These different preparations are from a gouty woman eighty-four years old, who died in the *Salpêtrière*, in 1863, and whose right hand is pictured in Fig. 2 of Plate I.)

FIG. 6.—Left ear of I. M——, an old coachman, born in Poland in 1807, and in whom the first attack of gout occurred at the age of twenty-five. (*Hôpital Rothschild;* service of Dr. Worms.)

h, h, h.—Large concretions of urate of soda. These tophi, so the patient says, commenced to appear three years after the first attack of articular gout.

Plate III.

Fig. 1.

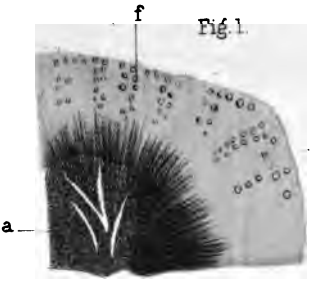


Fig. 2.

Fig. 3.

Fig. 4.

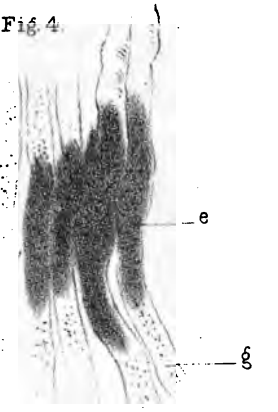


Fig. 5.

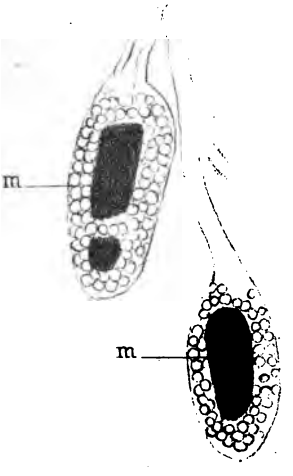
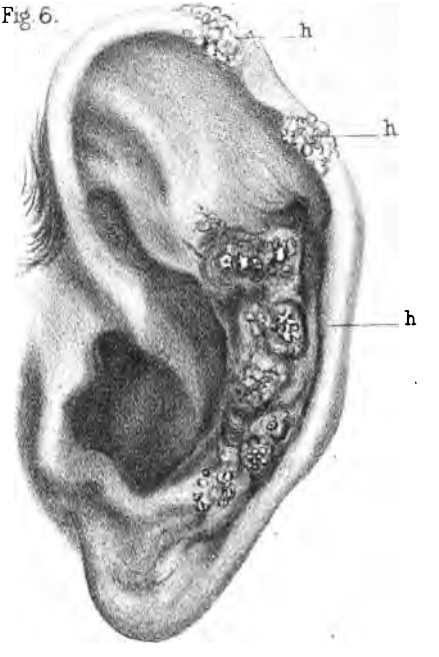


Fig. 6.



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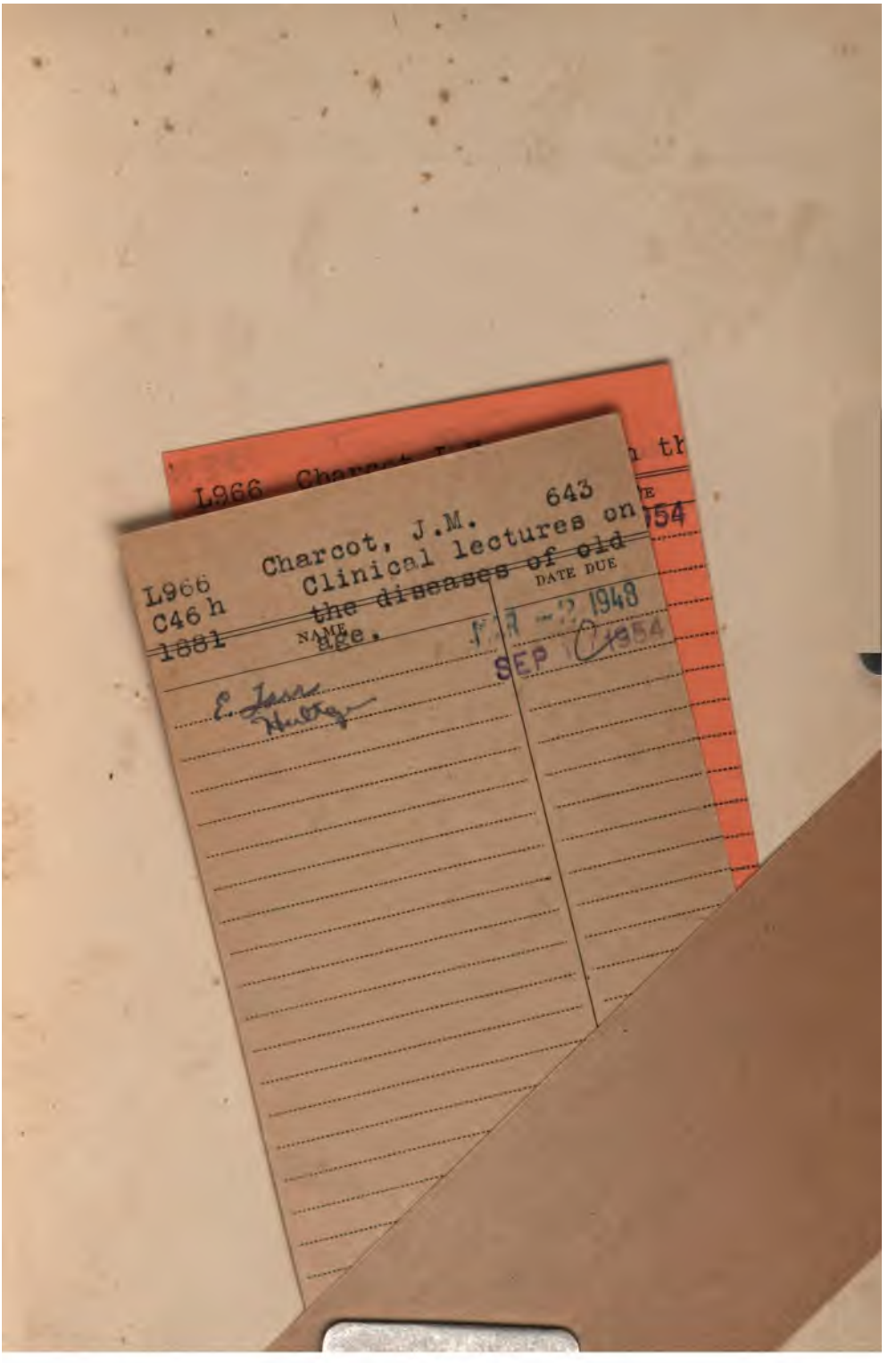
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